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XXXV

ON THE DIRECTION OF RESEARCH AS TO THE ANALYSIS OF CORTICAL STIGMATA AND FOCAL LESIONS IN CERTAIN PSYCHOSES

BY

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ON THE DIRECTION OF RESEARCH AS TO THE ANALYSIS OF CORTICAL STIGMATA AND FOCAL LESIONS IN CERTAIN PSYCHOSES[1]

By E. E. SOUTHARD, M.D.
OF BOSTON

I DESIRE to bring before this Association certain points and viewpoints in theoretical psychiatry, trusting to be able to persuade the Association members that the task of finding genetic factors in psychiatry is not absolutely hopeless. I know that psychiatry and, indeed, the entire field of neuropathology in its most general sense are looked on as hardly germane to practical medicine. Yet the members of this Association, while not willing to go on the witness-stand (as did the Philadelphian of legend) as *general specialists*, should be among those who would see the general connections of psychiatry most clearly.

Since 1906 I have been making, collecting, and tabulating observations, largely from postmortem data, which bear on theoretical psychiatry and more especially on genetic factors.[1] Naturally my data have been largely somatic, both neural and non-neural, although by turning special attention to cases without obvious brain lesions I have latterly developed some hypotheses about the so-called functional psychoses. [2] [3] [4] [5]

After paying some attention to the frankly and admittedly "organic" psychoses (general paresis[6] and other syphilitic mental

[1] Being contributions of the State Board of Insanity No. 42 (1915.8).
Bibliographical note: The previous contribution (1915.7) was by Rose S. Hardwick, entitled "Programs and Directions for the Mental Examination of Asocial, Psychopathic, and Doubtful Subjects," to be published in the Boston Medical and Surgical Journal, 1915.

diseases, and arteriosclerotic and senile atrophic conditions),[7] [8] I found most of my research interest passing to the region of the so-called functional psychoses, viz., to dementia precox[9] [10] (which, to be sure, is now beginning to be regarded in many quarters as "organic" in some sense) and to manic-depressive insanity[11] (about whose anatomy Kraepelin could say, in 1913, "there are no certain data"). I entered the investigation of these functional psychoses with the definite prejudice that nothing relevant would be found anatomically in the brains—that is, no evidence of lesion entailing permanent damage and cell-loss. Perhaps prejudice is too harsh a word for such a method of approach, since I hold that a brain, like an alleged criminal, should be considered normal (that is, innocent of lesions) until proved not so.

However that may be, the brains of dementia precox subjects, selected at random and examined in sufficiently large number, proved to exhibit certain anatomical appearances of a very intriguing nature. These appearances may perhaps be called lesions if the term lesion can be used to include both acquired lesions and the results of germ-plasm taint, embryonic and fetal factors, and such early acquired lesions as seem almost tantamount to antenatal lesions. But these lesions and stigmata, occurring in a large majority of cases of dementia precox (the larger, the more careful the analysis, as I have since shown by systematic photographic analysis), are exceedingly hard to analyze as to their genetic significance. On the whole I was impressed with their large significance as indicators of hereditary, congenital, or acquired weak places in the brain structure, finding as I did histologically that whereas the construction of the tissue might be faulty (anomalous architecture) there was superadded evidence of progressive disease entailing cell-loss and characteristic tissue reactions (scavenger functions of Bevan Lewis, supportive neuroglia proliferations of Weigert, etc.).

But, while the significance of such lesions as indicators of past and present disease must be provisionally accepted, it is not so clear that the particular symptoms of dementia precox can be safely correlated with the particular lesions found. Indeed, I suppose it would be unwise to count all dementia precox phenomena as essentially paralytic or postparalytic phenomena (in the sense of Hugh-

lings-Jackson), and dependent upon fine cell-losses, in that it was not likely that the principles of the old extirpation-neurology even when extended to finer anatomy, were to carry us far into the field of essentially irritative phenomena which so many mental symptoms and conditions assuredly are. Meantime, I have made certain correlations between site of lesions and character of symptoms that seem to me to hang together (frontal lobe lesions and delusions of ordinary type,[9] [10] [12] parietal lesions and catatonia,[9] [10] temporal lesions and auditory hallucinosis[10]). My argument does not here concern these correlations, although the cases mentioned below will involve some of them.

Restricting inquiry to the nature of such lesions and not dealing with their effects, let us ask the more general question, What is the relation of these lesions to dementia precox in general? Before approaching this question in detail, we must ask whether such lesions are found in other diseases. Do not normal persons possess them? And, even if they do not show acquired lesions superadded, may they not show anomalies of such sort and with such frequency as to make such lesions quite non-characteristic for dementia precox? It is curiously hard to answer this question and, from the literature, almost impossible. I have data partly analyzed from general hospital autopsy series from which I believe I am safe in asserting that no great number of the brains of sane persons possess such anomalies. But a more positive answer, and one to my mind quite convincing, is derived from an exact analysis of the brains of subjects of that other functional psychosis mentioned above, viz., manic-depressive insanity.

Here I find, instead of four brains out of five showing coarse evidence of brain-disorder (as in dementia precox), but one brain in five showing such brain-disorder in manic-depressive insanity. (Let me insist that the criteria were the same for both series and that the confusing issues of arteriosclerosis were excluded to the same degree from both series by leaving out cases with lacunar lesions due to vascular plugging.)

These two sister affections, dementia precox and manic-depressive insanity, in fine stand upon entirely different bases with respect at least to the gross visible and tangible effects of cell-loss. And

these findings are to be held as roughly correlatable with the different curabilities of the two affections, since the outcome of dementia in dementia precox is held to be characteristic and the tendency to recovery or to restoration of a symptomless condition is characteristic of manic-depressive insanity.

It is perhaps necessary to remark that these anatomical observations do not necessarily imply that the microscope will not discover lesions (and genetically important lesions) not related to anomalies and not issuing in sclerosis or atrophy. All we can say is that such lesions are unlikely to be anything but what logicians might call reversible; they would be lesions unlikely to result in cell-losses of any great proportion.

If I am asked why my figures are not absolute, why the fifth dementia precox brain does not show gross lesions and why the fifth manic-depressive brain does show gross lesions, I have several pertinent answers. (1) Our clinical diagnoses are not accurate to 100 per cent., although they possibly should be more accurate than 80 per cent. (Even the clinical accuracy of diagnosis in general paresis did not surpass 85 to 90 per cent. until very recent years, and with diagnostic devices nowhere approached in delicacy in the field here in question.) (2) Our anatomical diagnoses, especially those embodied in our protocols, are not always accurate. This has been emphasized in my recent photographic studies, wherein important anomalies are repeatedly shown which had been quite overlooked on the autopsy table. (3) Our conceptions of the two diseases are not entirely clear. Sometimes I get the impression that the distinctions drawn are to some extent upon false lines, although I do not doubt on present evidence that Kraepelin has arrived at a rough approximation to the truth.

Again, the microscope may come to our aid. With respect to dementia precox, I have recently found two cases of very brief duration in which elaborate photographic analyses of the gyri yielded no sign of gross lesion, but microscopically there was the most abundant and convincing evidence of neuroglia proliferation and cell-pictures decisive of the fact of cortical disease. Was there in these cases no long-standing cortical anomaly, and, had the cases lived, was the disease process finally to discover weak places in

the cortex such that topical scleroses and atrophies would supervene? or were these brains in process of a generalized mild atrophy? *Quien sabe?*

With respect to manic-depressive insanity, we are to some extent, aided by the current view that all of the characteristic clinical phenomena of manic-depressive insanity may occur in virtually any other form of insanity, since the manic-depressive phenomena are really only quantitatively different from the reactions of the normal man. If this is the case, we might easily believe that a case with a brain marked by an anomaly signifying a potentially weak place could still have symptoms such as a manic-depressive would have. Hence, a few cases might come through to autopsy with anomaly-bearing brains but with symptoms not at all related thereto.

However this may be, the fact of these differences remains in the tale of gross lesions which we can collect from these two sister affections. No one can deny, except on the basis of more penetrative analyses than my own, that dementia precox brains look abnormal much more often than do manic-depressive brains. But are these abnormalities inborn or acquired? And, even if not inborn, are some of them perhaps acquired so early that they interrupted development seriously at a period long prior to the appearances of symptoms? Are they, in short, anatomical appearances running *pari passu* with the course of symptoms, or are they rather *nidi* for the incubation of symptoms.

This is a question to which no general answer could or should be given. In the first place it cannot be said that either dementia precox or manic-depressive insanity is an absolutely well-established entity. In fact the entifying of these conditions will largely depend upon dicta of the postmortem room, the microscope, and the test-tube.

There are three sets of microscopic data which have attracted attention in analyses of dementia precox brains. There are (*a*) data concerning products of degeneration inside and outside of neurones.[13] There are (*b*) data concerning neuronophagia or satellitosis.[14] And there are (*c*) data concerning cell-losses. Probably the best-known work, at least with the two former groups of data, is that of Alzheimer, and it may be surmised that the data concerning

cell-losses given in Kraepelin's systematic account of dementia precox[15] (1913) are largely, if not entirely, based on descriptions by the same esteemed master of neuropathology.[16] Those of us who have enjoyed more or less work in Heidelberg or Munich are also aware how far these developments have been favored by Nissl.

The Nissl-Alzheimer school has, however, not made the same progress with dementia precox as with general paresis, largely, I suppose, for the technical reason that no such almost pathognomonic cell as the plasma-cell in paresis has been found to aid the tyro in the histological diagnosis of dementia precox. It was as early as 1897 that Alzheimer first called attention to the phenomenon now known conveniently as satellitosis in the cerebral cortex of catatonia.[14] That proliferation of neuroglia cells, especially in the lower layers of the cortex, had been thus found, was the chief contention of structuralists for their view of dementia precox. But with the lapse of time this contention has lost some of its force, partly because many cases failed or were said to fail to show satellitosis, partly because interest shifted to other matters (especially degeneration products), and partly because the conclusion has apparently been arrived at that infrastellate (lower-layer) gliosis is not so characteristic as suprastellate (upper-layer) cell-losses in the cortex of dementia precox.[15]

Accordingly, whereas the Heidelberg and Munich workers have persistently clung to a structural theory of the pathology of dementia precox, yet it cannot be said that they have clung to it consistently, as the basis of the claim has now been satellitosis, then *Abbauprodukte*, and again cell-losses.

Not being myself able to see anything differential in these different findings, I chose another line of attack and made careful topical examinations of the cerebral cortex in dementia precox,[9] hoping to show topical variations in the degree of whatever non-differential cell-changes might be going on and thus to determine the areas that suffered most. My efforts struck me as having a certain success. And I note that Kraepelin, whose structural notes for the volume published in 1913 evidently did not contain a reference to my work of 1910, has approximated my conclusions of 1910, basing his own upon an analysis of cases published by various hands.[16]

I do not, however, feel that enough work has been done to render any of these conclusions absolutely safe.

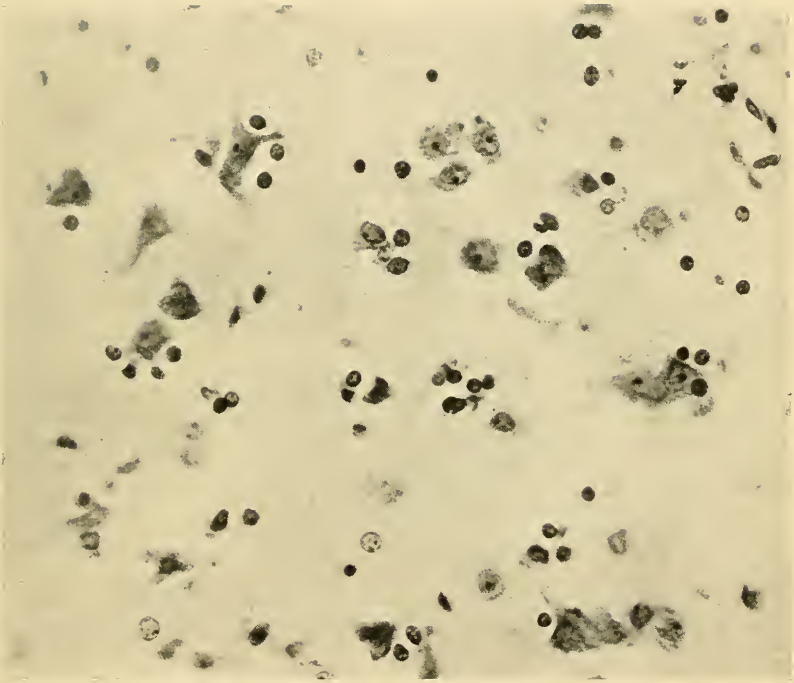
Tentatively in my own work I regard the degeneration-products of Alzheimer, or Marchi degeneration-products (non-Wallerian forms), or the iron-hematoxylin-staining substances with which I have personally worked,[7] [17] [18] as decidedly not differential for dementia precox. I understand Alzheimer to maintain that somewhat the same picture can be got in the tissues of a case of toxic delirium as in those of a catatonic *Hirntod*. The time relations of the appearance, fastigium and disappearance of these degenerative products remain to be established.

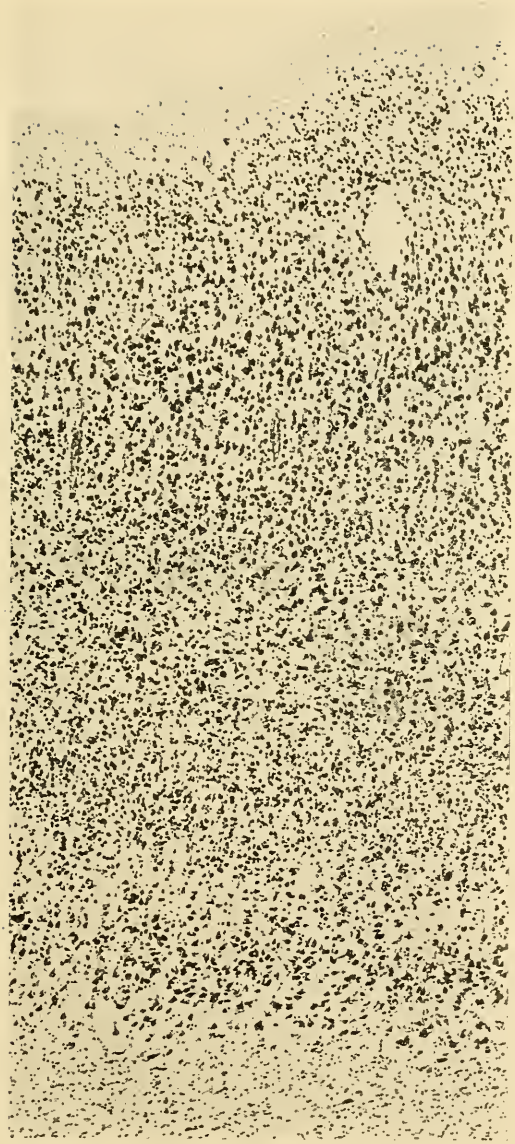
Satellitosis I find myself regarding as equally non-differential and as developing somewhat more slowly and at all events lasting much longer than the *Abbauprodukte*. There is, I believe, doubt in some minds whether the appearances of satellitosis are ever erased. Some unpublished observations which I have been making with experimental monkey poliomyelitis material indicate that satellitosis may disappear within measurable periods. But it is clear that satellitosis remains a striking phenomenon far into the period of demonstrable cell-loss, and it is probable that the phenomenon is closely related with some of these cell-losses, whether as furnishing an active agent of destruction or as responding to chemical stimuli from the dying cells.

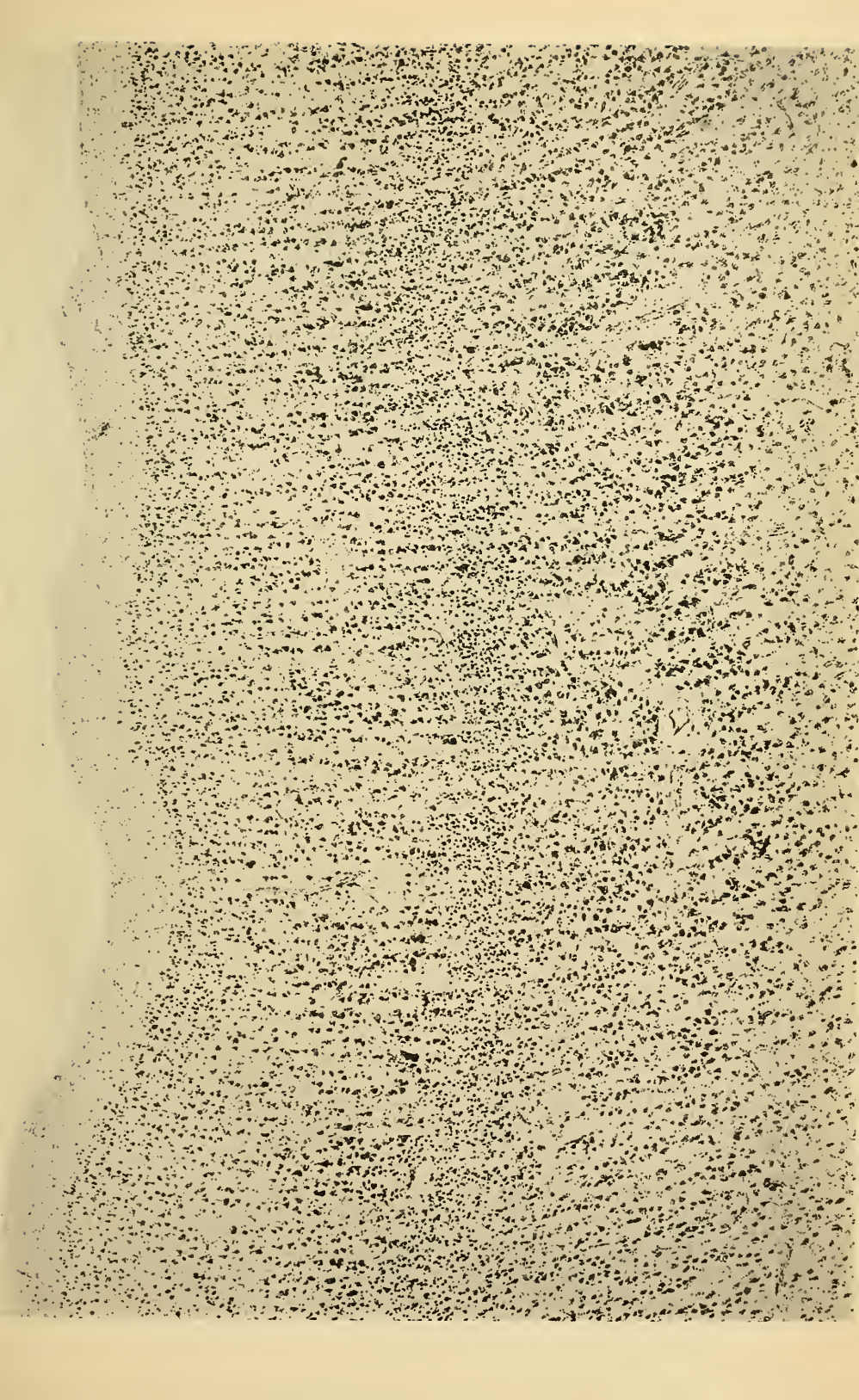
These three characteristic appearances, then, may perhaps be regarded as representing roughly successive histopathological phases in various degenerative diseases; but these phases may overlap and indeed be all present at the same time. But, from a combination of the phenomena, tentative conclusions can be drawn as to the order of events. Probably the least reliable of these sets of data is the degeneration-products, since it would appear that it is signally difficult to exclude terminal intoxications of one sort or other in cases dead of dementia precox or of any other mental disease. I have even been at some pains to show the extent of these possibilities in systematic cultivations of bacteria from the cerebrospinal fluid in several hundred cases.[19] [20] [21] [22] [23] In the absence of satellitosis and cell-loss the degeneration products taken by themselves would seem to be open to suspicion, unless they are extremely

DESCRIPTION OF PLATE I.

Plate I illustrates cellular gliosis, in this instance chiefly *satellitosis*, *i. e.*, proliferation of those non-fibril producing neuroglia cells found normally in small numbers (usually not more than a pair) next to certain large nerve cells. Specimen from infrastellate layers of postcentral gyrus in a case thought to be dementia precox, dead at seventeen after two months' symptoms. There was no gross lesion of the brain in this case.







marked in a bacteriologically negative case such as that reported by my colleague Orton.[23] In any event it is probable that the soaking of tissues with *Abbauprodukte* is to some, or even to a large extent usually a reversible process, and that after a time little or no more evidence of such deposits can be seen than of the pneumonic exudate after recovery.

In order to bring to a point my tentative stand toward the matter of satellitosis and of cell-loss and toward their significance, I wish to present samples of histological data from two cases. It would appear that the considerations turning upon these may prove of still greater value in future analysis of similar material of which I now have a large collection.

I choose two areas of the cortex of which we know comparatively a good deal—the occipital region and the post-central gyrus.

To illustrate a point concerning the occipital region, I take a case already reported[9] (as Case VI). I shall not here repeat the facts concerning this case other than to say that the patient died at the age of fifty-seven years, after perhaps thirty-two years of a mental disease regarded as the paranoid type of dementia precox, in which disease there were a number of striking features. It is noteworthy that the brain, which weighed 1490 grams at death, showed no signs of atrophy to the naked eye, although there seemed to be tangible evidence of induration of the brain substance so that on the autopsy table the diagnosis of generalized cerebral sclerosis was made. It was also noted—and this is particularly striking in connection with the findings microscopically—that the occipital lobes felt firmer than the rest of the brain. There was nowhere any evidence of chronic change in the pia mater except over the superior frontal gyrus of each side. Microscopically, it may be noted in passing that we found a considerable degree of change in numerous areas. Particularly under the thickened pia mater of the frontal region there was evidence of fibrillar gliosis. Incidentally, there was found a gliosis of the cornu ammonis (it appears that there was one episode of olfactory and gustatory hallucinations of an unpleasant character; these were also associated with visual hallucinations, also unpleasant).

The most striking thing in the case was the patient's description

of peculiar spiritualistic phenomena, with "forerunners and communications." I take the following from the description:

> Patient would often communicate with God, assuming a rigid position, gazing into space, and saying, "God, our Heavenly Father, I would like to communicate, if convenient, don't discommode yourself?" He pauses a few moments and then says "Is that you? Is that really you, Heavenly Father? Please come as an apparition, as strong as you can;" then in a faint voice, "That's pretty good, pretty good. Can you come a little stronger, I wish you would if you could; that's pretty good, that's good. God, Heavenly Father, is it safe to show the forerunner in the face of Dr. Mitchell? Are you sure, perfectly sure? All right." He would then make a peculiar movement of the hands which he alleges is a forerunner and has the power of destroying life. Asked to forecast the future, he calls up God as previously and says, "I wish to ask a question in regard to Dr. Mitchell's success in the future? Is your time precious, God? Can you spare it?" He turns to examiner and says, "He is gone now to examine. He explains by astronomy." In the meantime, patient converses pleasantly about the weather until God calls, and later assures examiner that his success is to be unlimited and that he has received communication. Result of these communications was, as a rule, optimistic.

Of course, it is *a priori* most unlikely that any correlation could be found between the specific content of combined and, as it were, *scenic* hallucinations of this type with microscopic findings. It is nevertheless, very striking that the tangible evidence of gliosis found in the gross brain was most marked in the occipital area. Accordingly, I examined with considerable interest the tissues from this region. It is, of course, true that our methods of gauging the amount of gliosis are still in their infancy. What I have been tempted to trust to, rather than the finer methods of very recent years, is evidence of neuroglia cell increase or such highly marked nerve cell loss that no one could fail to be convinced by evidence offered. *Satellitosis*, that is, proliferation of non-fibril producing so-called satellite neuroglia cells (found normally in small numbers, as a rule not more than two, at the base of certain nerve cells) is a fairly striking phenomenon when exhibited as in Plate I. (See description of plates.)

There remains some doubt what satellitosis, or cellular gliosis, of this particular type actually means. Satellitosis signifies in part

at least what the older term *neuronophagia* was intended to mean, except that the term *satellitosis* does not indicate that the cells are engaged necessarily in a phagocytic process, or are in any sense actively devouring fragments or products of nerve cells. Nevertheless the satellites proliferate very probably under definite chemical conditions next to the nerve cells, and it is the belief of some authors that this neuroglia cell proliferation is an index of a degree of cell or tissue loss.

To return to the case of dementia precox just cited as having peculiar spiritualistic phenomena. A microscopic examination of the occipital cortex must consider both the type of cortex called "calcarine" and found in association with the white line of Gennari (or of Vicq d'Azyr), and the type of cortex which may be termed "common occipital," which surrounds the calcarine type, lying in general forward thereof. It is true that the mapping of this region by the topographers, Bolton, Campbell, Brodmann, and others, has not definitely decided what the still further anterior limits of the common occipital type of cortex are. But from the present more modest stand-point of histopathological analysis, it suffices to consider that there are very definite and easily observable distinctions between the two types of cortex which will readily permit us to draw conclusions as to the differential nature of lesions found in the two cortex types. In Plate II, a portion of the calcarine cortex is displayed which may not be in all respects normal, but at any rate fails to show evidence of cell loss, and with a hand lens one fails to discover convincing evidence of satellitosis such as that shown (with higher powers of the microscope) in Plate I.

In Plate III, however, from the same case, and from the same block of tissue fixed and prepared by the same technique, observation with the hand lens will readily show in the lower part of the photograph numerous neuroglia cells, many of them in close relation to nerve cells. Most of the cell increase here shown is in the infrastellate layers, namely: in the lower half of the section as photographed; but traces of the change are also to be found in the suprastellate layers.

The argument from a comparison of appearances in Plates II and III is plain. Disregarding for the moment whether either

DESCRIPTION OF PLATES II AND III

Plates II and III are from a single microscopic preparation (alcohol fixation, original Nissl method, unimbedded) of the occipital cortex of the "spiritualist" victim of dementia precox with recurrent semivoluntary visual hallucinations. Plate II illustrates the calcarine or "visuosensory" (Bolton) area and does *not* show satellitosis. Plate III (from common occipital or so-called "visuopsychic" area) shows well-marked satellitosis (of general type resembling that shown in high power in Plate I), especially in the lower or infrastellate layers. There is also demonstrated the normal sharp differentiation between "visuosensory" and "visuopsychic" cortex. The pathological point turns on the satellitosis confined to the "psychic" area.

area is entirely normal in all other respects, it is certainly clear that the two areas are different in their content of satellite and neuroglia cells. I will not stop to inquire, moreover, into the question of the original quota of neuroglia cells in the two types or cortex. Future research may show that the common occipital type of cortex is better prepared with neuroglia cells than is the calcarine type. Even if this be true, it is still plain that the two regions must differ in their histopathological reaction to agents of like nature.

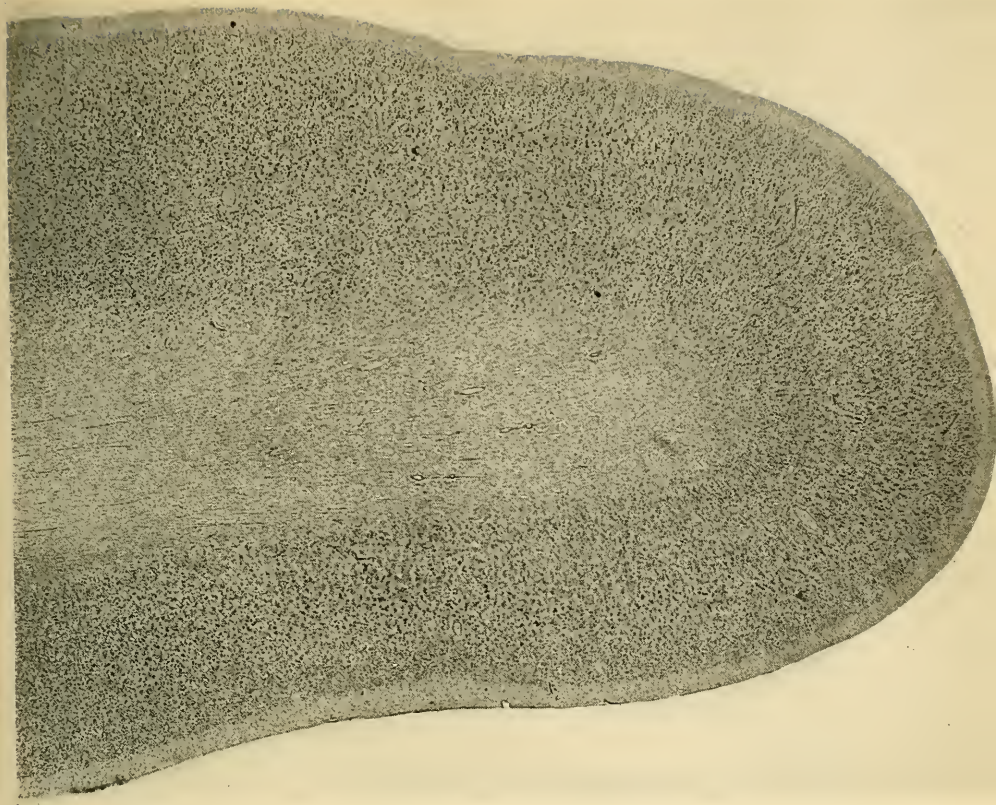
If I now add that the occipital pole is perhaps better understood than any other single area of the cerebral cortex with respect to its coarse functional relations, namely, the higher processes of vision, and if I add that all the work of the topographers above mentioned seems to show that the calcarine area is more of an arrival platform for impulses coming directly up from the thalamus and similar inferior sources, whereas the common occipital type of cortex is very probably more of an elaborative, associative, and combining nature, then I shall have made clear the general trend of my argument. If the visuo-sensory (calcarine) area has one sort of histopathological tendency, and the visuo-psychic (common occipital) type of area has another sort of reactive power, then it is clear that a toxin or chemical or physical, or other condition acting equally upon the two areas is nevertheless very likely to produce unequal results therein.

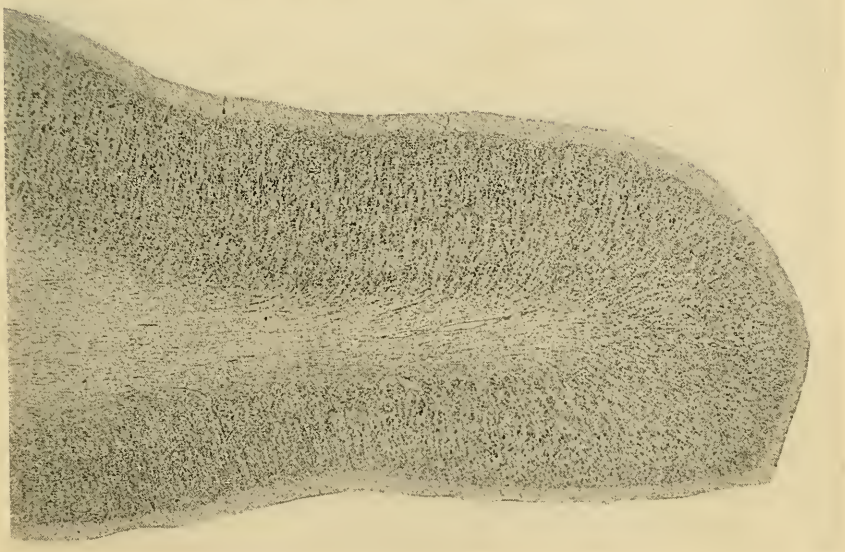
To sum up regarding the case of the spiritualist, it is unlikely that the appearances in Plate III are precisely responsible in any sense for the "forerunners and communications" of the case. But it is clear that by such methods, we shall be able to get a much more intimate insight into special conditions of the cortex and to discover special labilities of particular areas. As in future, physiology, comparative anatomy, and pathology, throw more light on special functions of areas, the meanings of these special liabilities of different cortex types will become clearer and clearer.

I now proceed to the discussion of a less well understood area, namely, the postcentral gyrus. To be sure, concerning the postcentral gyrus, we all remember the striking demonstration of Sherrington, who proved conclusively that the electric excitability

DESCRIPTION OF PLATES IV AND V

Plates IV and V contrast with low magnification the appearances in coordinate portions of the left (larger) and right (smaller) postcentral gyri in a case of catatonic dementia precox, described in the text. The inequality is striking enough, but each gyrus preserves certain normal features, *e. g.*, the shallower depth of that portion of the gyrus which faces anteriorly. We are here dealing perhaps in large part with a cerebral anomaly.





of the so-called central area was not a property of the postcentral gyrus, but was limited to the precentral gyrus. The general conclusions of Campbell concerning the histology of the gyrus must also be kept in mind, although the work of Holmes and of Brodmann has thrown some doubt upon the details of the gyrus. The question may perhaps remain unsettled, whether we shall count the postcentral gyrus as having two types—one corresponding to the anterior flank, and the other to the posterior—or whether we are to consider that the tissue at the crown of the gyrus represents a novel third type of cortex. Plates IV and V of the case which I shall discuss briefly below, show the range of variation in the cortex types on the sides and at the crown of the postcentral gyri. The anterior face of the postcentral gyrus is normally very thin, and it is clear that both the narrow and the broad postcentral gyri in the case about to be described show in characteristic form the lesser thickness of the cortex on the anterior face than that of the posterior face of this gyrus. It is in this region that Holmes apparently showed Campbell to be in error as to lesions which Campbell thought he had found in the postcentral gyrus of tabes dorsalis. Brodmann unconditionally states that Campbell's findings on the anterior face of the postcentral gyrus are nothing but normal appearances. Brodmann charges Campbell with ascribing pathological changes, cortical atrophy, and cell loss to an area which is in a perfectly normal state. Brodmann somewhat sarcastically says that the situation may be the same in numerous other pieces of work, and he thinks that the so-called pathological changes in the hippocampal region in epilepsy may be perhaps nothing but normal conditions.

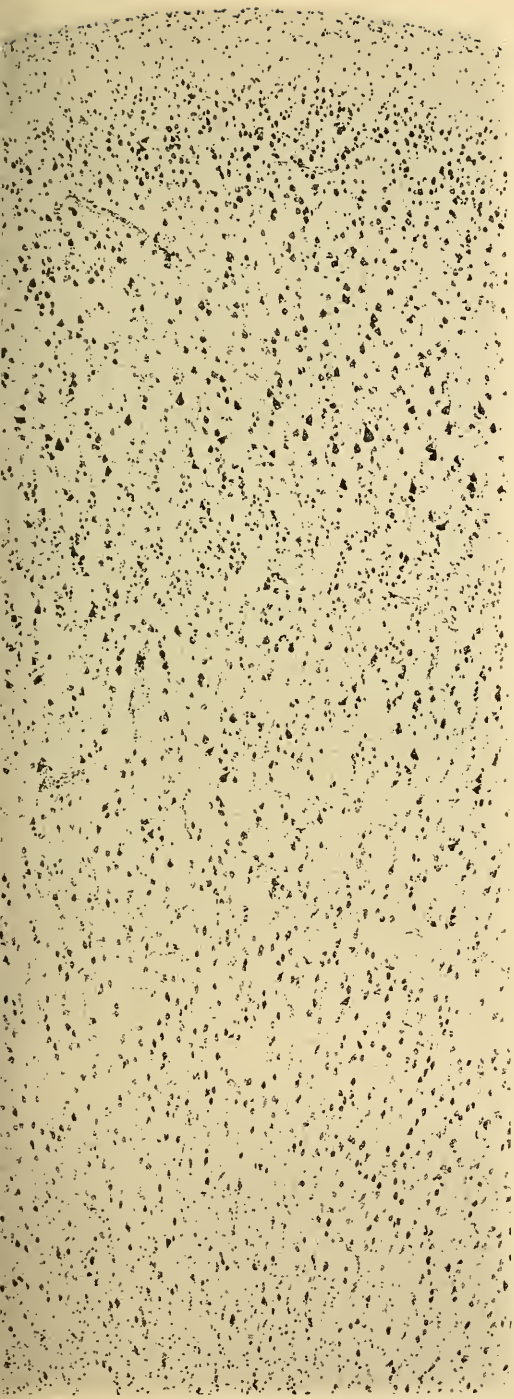
It is clear, however, that in a case like the one to be presented this difficulty does not hold. The reliance of the histopathologist must often be placed on a comparison of coördinate areas on the two sides of the brain. It seems to me that the appearances in Plates IV and V, showing two postcentral gyri in a case of dementia precox, exhibit clearly at least one point, namely: that the gyri differ in some respects at least. Whether the change represents an atrophy and a hypoplasia or a hypogenesia, is a question which cannot be settled without the most elaborate study.

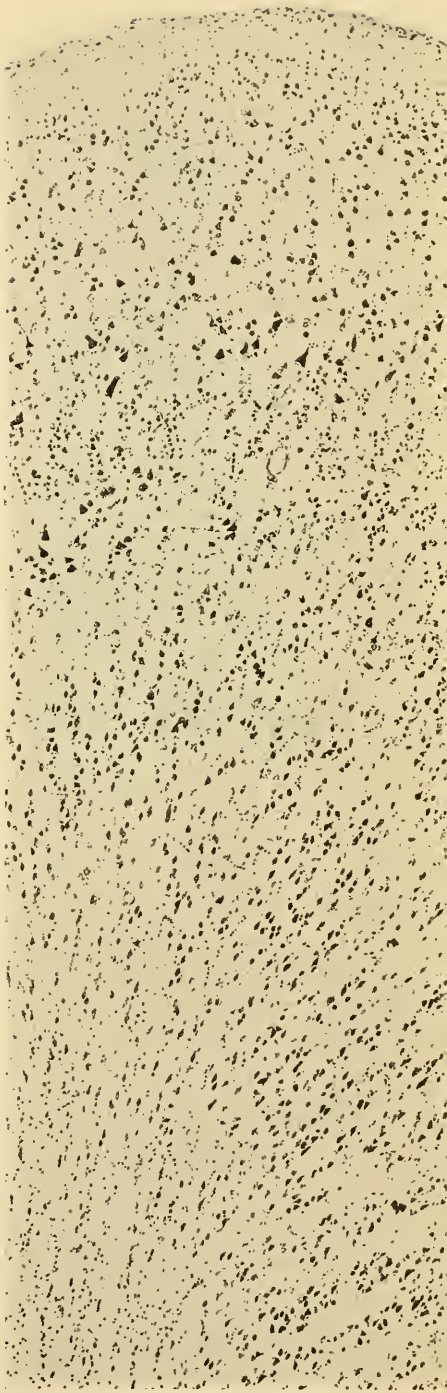
For the moment I desire to make but one point, namely: that we

DESCRIPTION OF PLATES VI TO XI

Plates VI to XI show appearances with higher magnifications from the gyri in catatonic dementia precox shown in low power in Plates IV and V.

Plates VI and VII contrast strips from the crown of the left (larger) and right (smaller) gyri, respectively. *Although it might have been supposed that the smaller gyrus would appear more diseased than the larger,* in point of fact far more satellitosis is discernible in Plate VI (from the "larger" and more grossly normal-looking gyrus). The point is, perhaps, that the right postcentral gyrus had long since passed through and by the phase of satellitosis (or, was it never capable of such reaction?). Of course, the small right gyrus does show relative poverty of *nerve* cells.





have two different conditions with respect to neuroglia proliferation held in the gyri; but before proceeding to speak of this, I wish to sum up the somewhat meagre clinical findings in the case, which has not hitherto been published. The data are as follows:

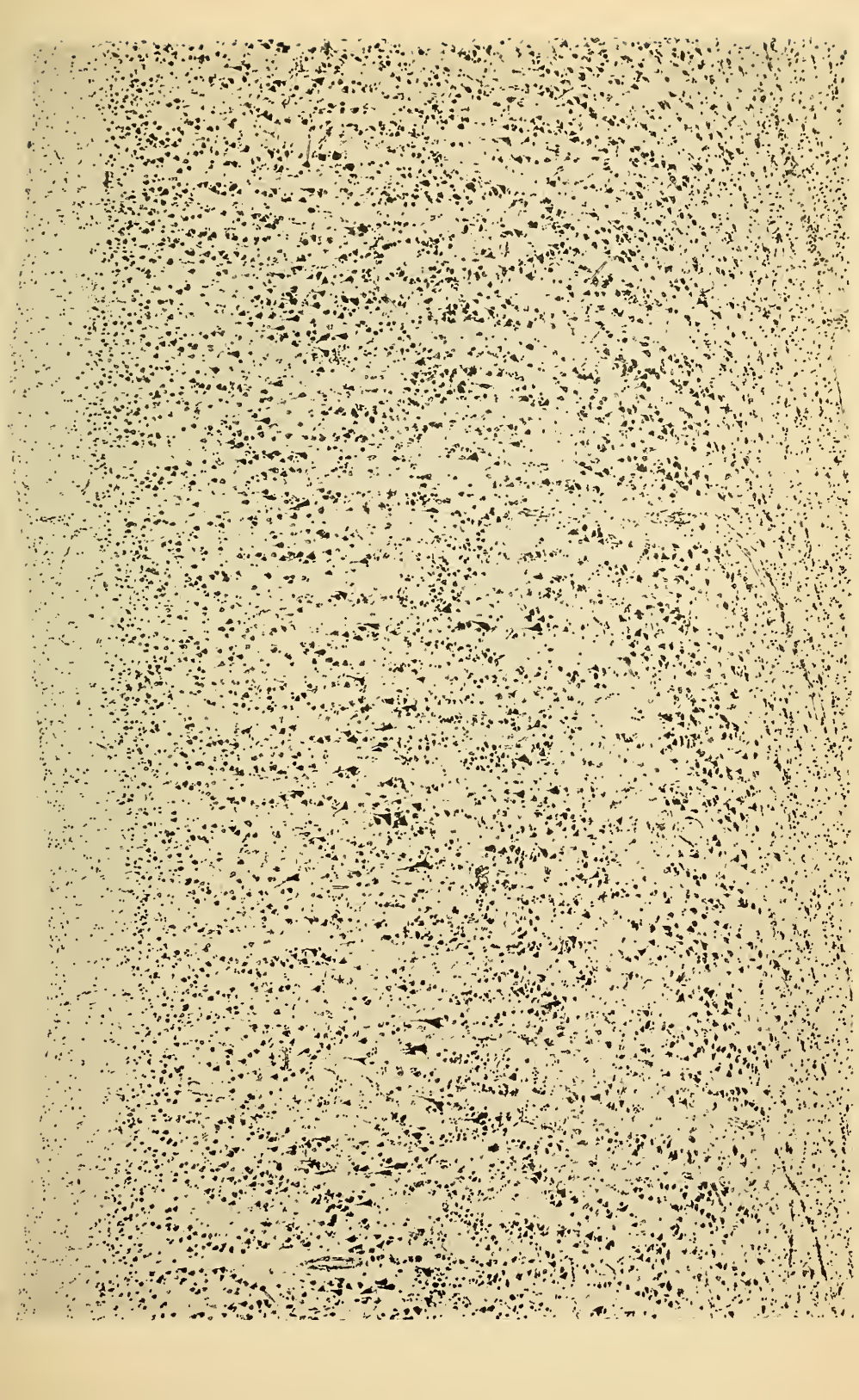
P. F., No. 8300, Danvers[8] Path. Lab., 1151. Married; housewife (sister insane) of Italo-American stock; had an attack of "confusion" in March, 1896, and then rapidly developed a violent acute mania, causing her commitment on the third day, June 22, 1896, at the age of twenty-seven years. The attack apparently began with confusion, mixing baby's milk (patient still lactating, but age of baby not known), followed by anorexia and insomnia, bewilderment, apprehensiveness, declamatory perseveration ("forgive and forget"), spells of hilarity, carelessness in appearance, untidiness. Patient improved in hospital but had spells of confusion and persisted in untidiness. Plunge baths failed to affect the condition. Desiccated thyroid (February, 1897) increased the pulse rate and markedly weakened the patient; but the mental state was improved and the patient became only occasionally untidy. March: stupor and spoon-feeding, untidiness; spells of excitement and glass-breaking. Then relapsed into a quiet apathy with excitement at menstrual periods. Periods of catalepsy (no details available); mutism with occasional laughter at things going on. Variable course, for the most part apathetic, but with spells of elation, singing, laughing, denudativeness, destructiveness, untidiness (June, 1900). Occasionally mute and resistive. In apathy sat in stereotyped position for hours, resisting efforts to change it. Walking with other patients would habitually keep hands over eyes. Periods, so characterized, alternated irregularly for several years. By 1905, the patient had become more antisocial, making violent attacks on patients, climbing on window sills and under tables. In 1907, the patient is described as keeping both hands over eyes for about a third of the time, always in one place in the ward, mute. Sudden transition to excited state, in which denudativeness and rolling about the floor became prominent features. Otitis media, constipation, general cutaneous hyperesthesia, and a peculiar hypertension of muscles described as "stiffening," without twitching or opisthotonos. Death after eight days, March 7, 1907.

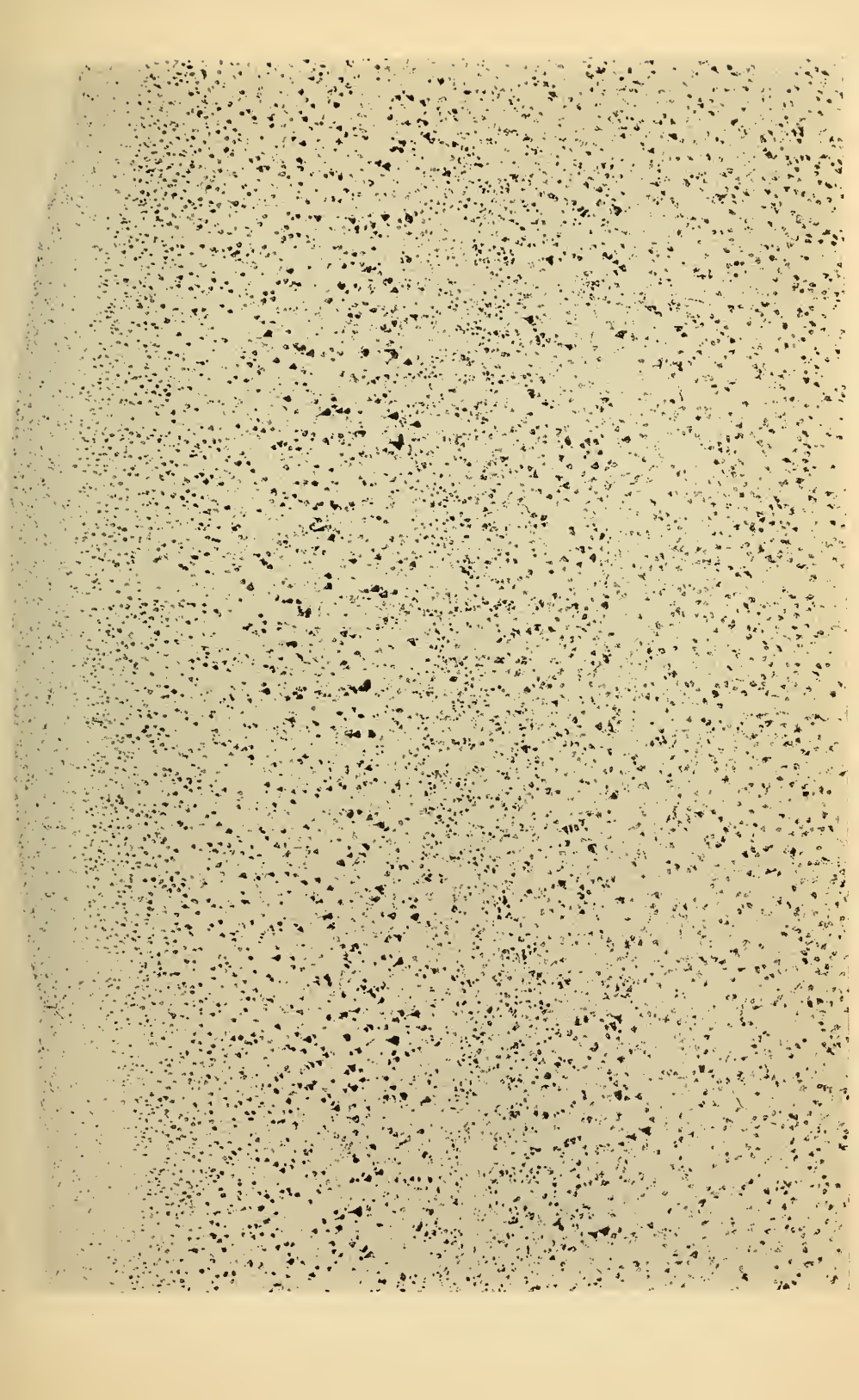
The findings in the head were as follows:

Calvarium not unusual; dura mater non-adherent; pia mater somewhat injected; basal vessels not thickened; brain weight 1045 grams; (by Tigges' formula, the brain weight in a subject 153 cm. long should have been 1224 grams); the cerebellum, pons, and medulla weighed together 150 grams.

DESCRIPTION OF PLATES VIII AND IX

Plates VIII and IX, similarly contrast strips from the posterior faces of the right (or small-looking) and the left (or normal-looking) gyrus. Possibly there is nerve-cell poverty in both gyri. There is far more evidence of satellitosis in the more normal-looking gyrus (compare statement under Plates V and VI).





The anatomical findings in general indicated either an atrophy or a hypoplasia. The general consistence of the brain was firm. (This might and probably does indicate gliosis, but gliosis is no proof that there was not originally a hypoplasia upon which the indurative process has supervened.)

The frontal convolutions were small; *the right postcentral convolution was about half the size of the left*; there were small cysts in the basal ganglia described as "superior to the knee of the internal capsule on both sides." The pia mater over the vermis of the cerebellum is described as "thickened and edematous." The cause of death was broncho-pneumonia with pleuritis, and there was also an acute dysentery.

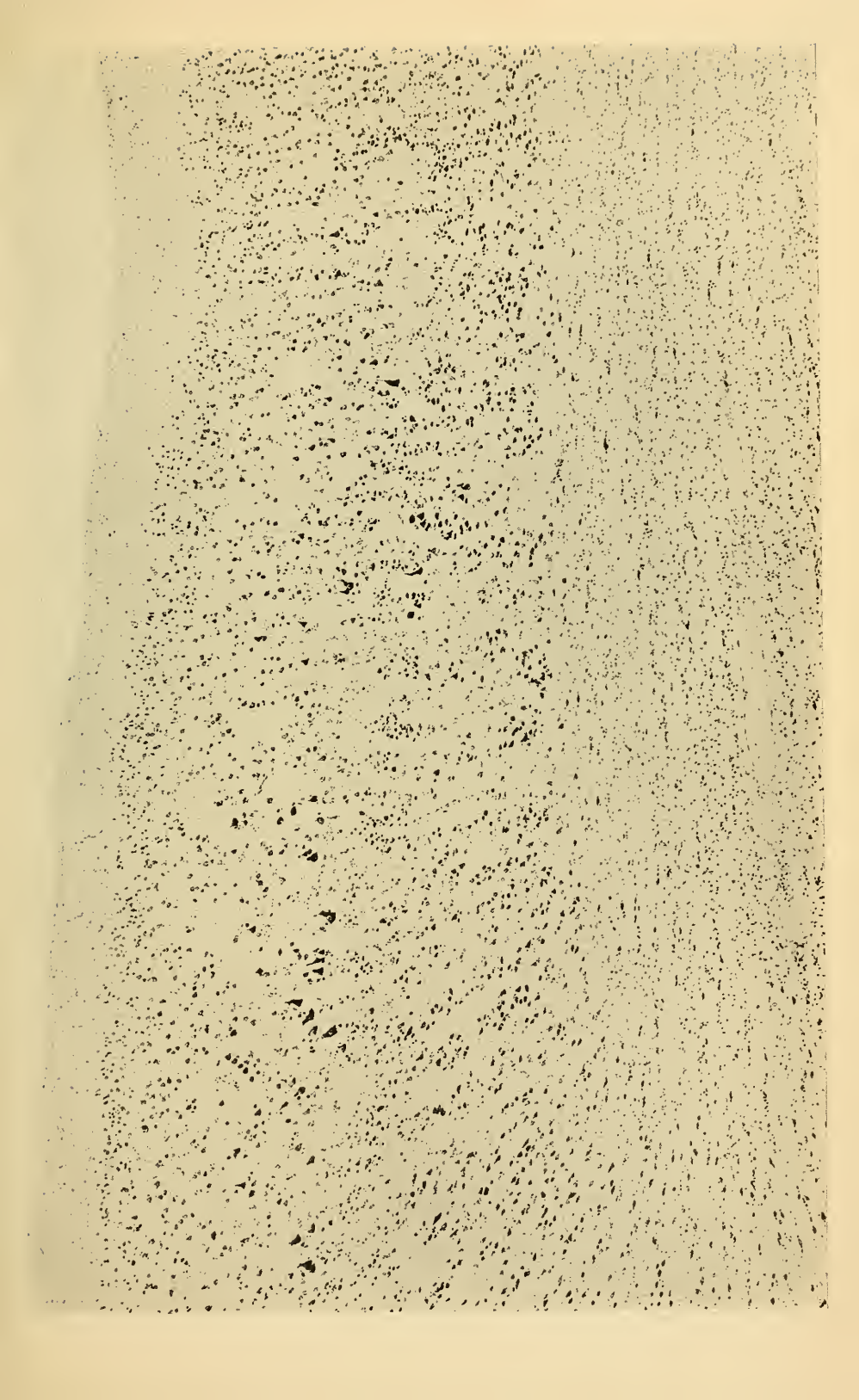
The kidneys showed a chronic diffuse process; there was a slight aortic sclerosis; there were a few milk patches on the pericardium; there was cholelithiasis and chronic splenitis, there was evidence of small old hemorrhages of the pelvic peritoneum and omentum; there was an erosion of the left acetabulum (with anomalous position of the head of the femur).

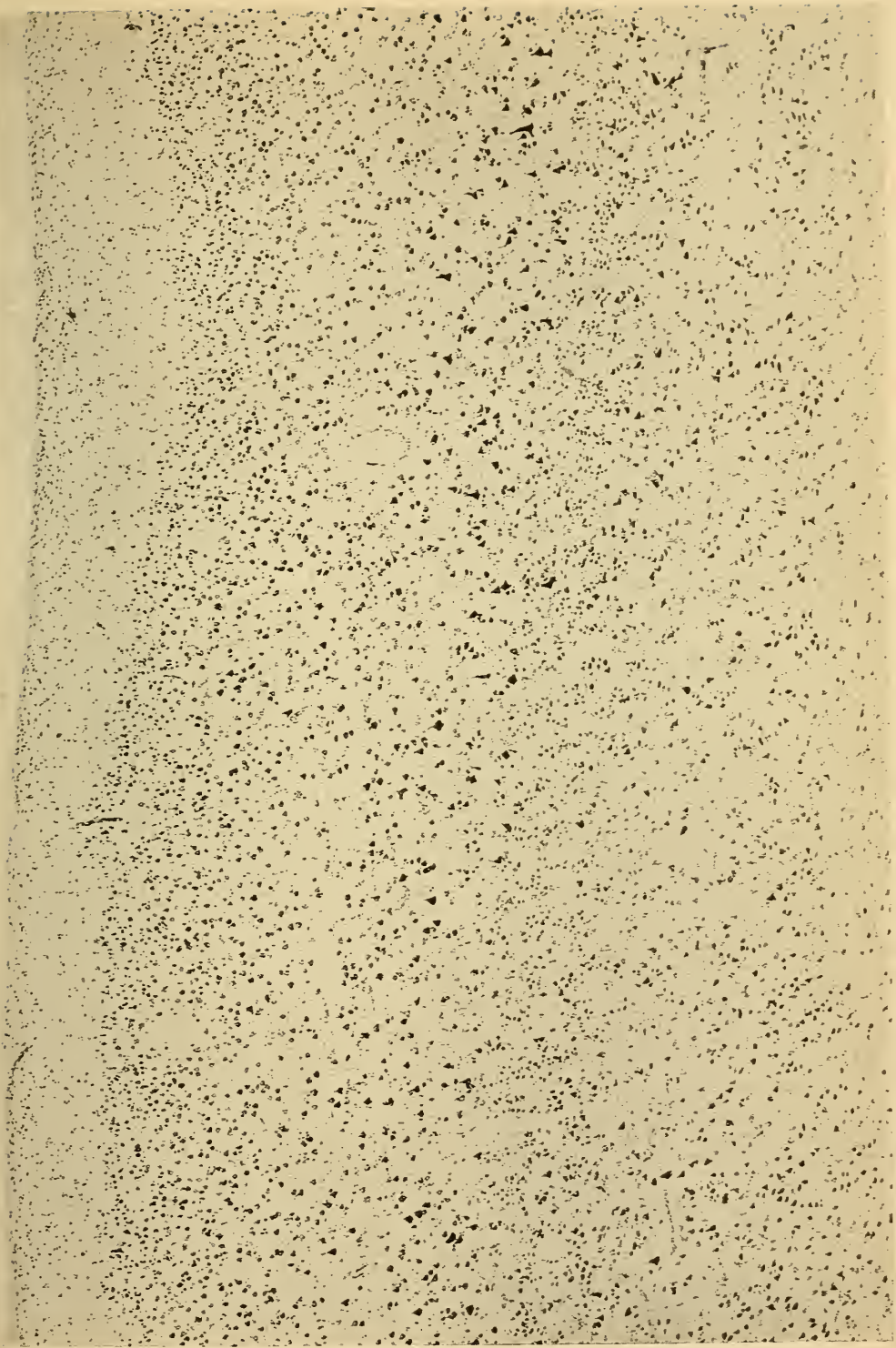
There is no need to discuss in this place the claims made by the writer as to the correlation of catatonia with post-Rolandic regions and of that exquisite phenomenon of catatonia known as cerea flexibitas with lesions of the postcentral gyrus. The present case was catatonic at times, and there appears to have been a "muscle stiffening" phenomenon, somewhat comparable to cerea flexibilitas. However, whatever the truth may be as to the correlation between the postcentral gyrus regions and muscular hypertension shown clinically, it is at least of importance to consider a fact in the histopathology. This fact is the rather striking fact that the best evidence of satellitosis is found, not in the narrow gyrus, but in the broad gyrus. Accordingly, if one were of a mind to regard the narrowing of the gyrus on the right side as due to an acquired disease process, one must consider that said process has been carried through to a virtual conclusion, and that the satellitosis and neuroglia cell proliferation which may have marked its course has disappeared. Further details are given in description of plates.

Summary and Conclusions.—In the above communication I have endeavored to bring out what I regard as an important line of structural research in mental disease. The general point of view on which I stand may be regarded as somatic although the topic which is most important for psychiatry is undoubtedly the functional psychoses (especially dementia precox and manic depressive psy-

DESCRIPTION OF PLATES X AND XI

Plates X and XI once more contrast strips from the two postcentral gyri (X from the right or anomalously small gyrus and XI from the left grossly normal-looking gyrus. Nerve-cell poverty is probably again in evidence in both gyri, although this condition is notoriously hard to diagnosticate. There is no important amount of satellitosis in either of these cortical strips (unless there is a trace on the left side, viz., in Plate XI). But perhaps none should be expected since the anterior face of the postcentral gyrus is doubtless far more sensory (analogous to calcarine occipital tissue) and the posterior face far more elaborative and, as it were, "psychic" (analogous to common occipital tissue).





chosis). In dementia precox I find four-fifths of the cases showing at autopsy certain appearances which may be regarded as anomalies or lesions in some sense. Possibly they should be regarded as *weak places* in the brain structure in which there may be later every evidence of progressive disease. The percentage of cases of dementia precox showing these lesions is 80 per cent. or higher. The percentage in the brains of non-psychopathic subjects has never been properly established, but in the so-called functional mental disease, manic depressive psychosis, I find similar anomalies or lesions in about 20 per cent. of all cases.

Accordingly, I hold that dementia precox is a disease in which cortical stigmata are much more often found than in certain other forms of mental disease and probably decidedly more often than in the normal citizens of the world.

The direction which research should take as to these findings is important. My point of view here is again a structural one. I present some photographs from two cases which indicate what I think will prove a rich line of research. There are two main lines of consideration.

First. We may study the appearances in those tissues which are regarded as the sensory arrival-platforms of the cerebral-cortex (for example, the calcarine type of occipital cortex) and contrast the findings in the sensory arrival-platforms with findings in the elaborative tissues which are adjacent thereto (for example, the common occipital type of cortex in the occipital region just mentioned). It is currently thought that we can safely call the calcarine type the visuo-sensory type and the common occipital type the visuo-psychic part of the cerebral-cortex. When we are able to get under the same cover-glass materials fixed, prepared and stained in the identical manner and observable in the same thickness, we are undoubtedly able to attach much consequence to the results of microscopic examination.

Secondly. We are able in certain cases to use the bilaterality of structures in the brain to help us in our interpretations. I present photographs in another case which illustrate the line which research may well take. One post-central gyrus in this case was about half the thickness of the other.

The interpretation of the cell richness, the possible cell losses and the nature and degree of neuroglia cell reaction is not as easy as might appear at first sight. Particularly important is the question of the form of neuroglia cell proliferation which is variously termed neuronophagia and satellitosis.

In the present argument I point out that the visuo-psychic tissues of a certain case showed satellitosis whereas the immediate adjacent visuo-sensory tissues failed to do so. On the other hand, I find that the narrow and apparently decidedly anomalous postcentral gyrus of another case fails to show satellitosis but that its fellow on the other side, showing no gross lesion, shows frank evidences of satellitosis when examined microscopically. The point perhaps is that the narrow gyrus has completed its pathological evolution and has passed the phase of satellitosis; but our results here must remain problematical until we know more as to the intimate nature of satellitosis.

My total argument for a certain optimism in structural research in psychiatry is accordingly founded, not upon the interesting clinical correlations of the two cases (*A*, striking scenic visual hallucinosis, satellitosis of common occipital cortex, *B*, catatonic phenomena and anomalies of the postcentral gyri), but rather upon the more general consideration that we now have, owing to the efforts of the modern cortex topographers, the basis for differential histopathological analysis of adjacent cortical tissues of different functional significance, and the benefit of examining tissues of coördinate nature on the two sides. The careful attention of the histopathologist in the nervous system should accordingly be given to all those planes in which arrival-platform tissue comes into contact with higher elaborative tissues in the sense of the modern cortex topographers; and the findings in any gyrus should be controlled by study of the corresponding gyrus of the other hemisphere.

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16. I have summarized Kraepelin's latest account in my paper. On the Topographical Distribution of Cortex Lesions and Anomalies in Dementia Precox with Some Account of their Functional Significance (see note 10).

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XXXVI

ANATOMICAL FINDINGS IN THE BRAINS OF MANIC-DEPRESSIVE SUBJECTS.*

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ABSTRACT.

I. *Introductory.*

Object of this work, to control the writer's dementia præcox work (1910).

Symptomatological value of mild localized lesions.

Microlocalization and macrolocalization.

Brain consistences specially studied.

Gliosis and brain consistence.

Manic depressive insanity and dementia præcox.

Prognosis and diagnosis.

Autopsy findings uncertain in manic-depressive insanity (Kraepelin).

Thalbitzer's suggestion.

Significance of focal scleroses in silent brain areas.

Paucity of brain material "normal" in all ways in manic-depressive insanity.

Disintegration products in brains of catatonic excitement (Alzheimer).

The distribution of these should be studied.

Orton's results in a study of satellitosis in manic-depressive insanity and other diseases.

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* This paper is Number 36 (1915, 2), Contributions of the Massachusetts Board of Insanity, and Number 54, Danvers State Hospital Contributions. Some of the conclusions were presented at the meeting of the New England Society of Psychiatry at Rutland, Mass., in September, 1909. An abstract was presented at the seventieth annual meeting of the American Medico-Psychological Association at Baltimore, May 26-29, 1914. (*Bibliographical Note.*—The previous contribution (1915, 1) by E. E. Southard and M. M. Canavan, entitled "A Study of Normal-looking Brains in Psychopathic Subjects: Third Note, Boston State Hospital," was published in the *Boston Medical and Surgical Journal,* Vol. CLXXII, No. 4, January 28, 1915, pages 124-131.)

II. *Analysis of Case-Groups, with Special Reference to Excluded Cases.*

Cases with focal lesions (anomalies, scleroses, atrophies) especially interesting in relation to dementia præcox.

49 cases in the original random group.

6 cases of involution-melancholia, excluded from prior consideration.

4 cases excluded as containing focal arteriosclerotic lesions, rendering analysis of other focal lesions difficult.

1 case excluded as frank error of diagnosis.

11 of remaining 38 (29 per cent) show lesions recalling those of dementia præcox; but of these 11, 3 seem (*post facto*) actually to have been cases of dementia præcox.

2 others are of very doubtful diagnosis and 2 more somewhat doubtful, leaving 4 focal-lesion cases in 31 cases of manic-depressive psychosis.

13 per cent focal lesions (anomalies, scleroses, atrophies) in manic-depressive psychoses may be pitted against 86 per cent such arrived at by a similar analysis of dementia-præcox material.

III. *Analysis of Case Material, with Special Reference to Cases Showing Anomalies, Sclerosis and Atrophies.*

Involution-melancholia excluded (6 in 49).

16 cases in 43 show gross lesions (4 of these arteriosclerotic lesions, leaving too complicated a picture for first analysis).

3 in the focal (*non-arteriosclerotic*) list of 12 should be considered cases of dementia præcox (analyses given).

2 others probably best excluded as involution-melancholia and dementia præcox respectively (analyses given).

IV. *Notes from the Literature on "Organic" Cases of Manic-Depressive Psychoses.*

Pilcz on periodic psychoses: emphasis on arteriosclerotic cases (excluded from the present analysis).

Pilcz' hypothesis (1901) of faulty construction of nervous system not upheld by F. Hoppe, 1908.

Other cases.

The literature points to the great importance of heredity, and brings up questions as to the relation of arteriosclerosis and dementia.

V. *Special Questions.*

Tabulation of data.

Example of a non-hereditary case (data not above reproach).

Is not manic-depressive insanity essentially hereditary, in the sense that near relatives invariably show signs of insanity?

This result probable, if involution melancholia, focal-lesion cases, and decidedly atypical cases be excluded.

Dementia in manic-depressive insanity not yet proved to be due to arteriosclerosis.

VI. *Conclusions.*

I. Introductory.

The first purpose of the present study was to effect a proper control of the writer's brain findings in dementia præcox (1910).[1] The intrinsic interest of findings in manic-depressive insanity, whether positive or negative, has prompted a more elaborate study than at first contemplated, and a photographic anatomical study is in preparation, following the lines of the writer's later study of dementia præcox (1914).[2]

By and large, the study of brain *anatomy* in the insane, as distinguished from *cortex histology,* has been much neglected of late years. While studying microscopic findings more or less intensively in these cases, I have relegated histology to the background so far as possible, both in the dementia præcox work already published and in the present work on manic-depressive insanity; and I have sought to emphasize the *possible symptomatological value of mild localized cortex lesions* in a manner which will recall the methods of Hughlings Jackson and Theodor Meynert, rather than the more modern intentions of exactness displayed in several works.

Meantime, I recognize that the task of future psychopathology will be tremendously cleared up by feats of *microlocalization* in the cortex, beside which the present efforts look small. What I seek is a rough orientation in this field, a kind of coarse *macrolocalization,* which is an indispensable preliminary to more finished work.

The suitability of my material for this study is enhanced by the fact that I had been for some time carrying on investigations of *soft brains or brains with soft spots* from a bacteriological point of view,[3 4 5] and other investigations of *hard brains or brains with hard spots,* more particularly in connection with the study of gliosis in epilepsy. The consequence was that my protocols and those of my colleagues were filled with data concerning topical variations in brain consistence, coupled with observations on visible atrophies, macrogyrias, or microgyrias, in a large group of cases. I am convinced that future brain anatomists should carefully consider these *tactile data* in addition to the classical data afforded by the eye. Weigert used to insist in his laboratory that the neuroglia method would yield neurological results often superior to those afforded by the myeline-sheath

method. It seems to me that palpation of brains, revealing varieties of consistence due to differences in the framework of the tissues, is the natural preliminary of such neuroglia studies and teaches us *where* to spend our best microscopic effort.

To the practical psychiatrist, manic-depressive insanity and dementia præcox are sister affections. Not merely is our knowledge of both diseases largely the product of Kraepelin's synthetic insight, but also in psychiatric practice these two affections, theoretically distinct, produce between them a perfect mare's-nest of diagnostic difficulty.

Practically, the alienist is much concerned over this distinction because the prognosis is often assumed to hang upon the diagnosis, and because it is well known that the direction and intensity of our treatment hang very much upon prognosis. There is no doubt that many less strict diagnosticians, in America at least, proceed on the practical basis that manic-depressives may get well, but primary dements do not. Everyone knows somewhat successful practitioners who make up a prognosis in some cryptic, not to say feline, manner, and then proceed to label the case " M. D." or " D. P." according to the supposed favorable or unfavorable outcome. We are all acquainted, too, with eager psychiatric critics, who triumphantly demonstrate, months after some ardent staff controversy, that such a case was not " M. D." because it deteriorated, or was not " D. P." because it recovered. I need scarcely recall that Kraepelin himself lays down no such hard-and-fast lines of prognosis.

With respect to autopsy findings, Kraepelin himself dismisses the subject (1904) with these words (after the section on maniacal conditions): *" Von irgend gesicherten Leichenbefunden ist noch nichts zu berichten."* [6] The same sentence stands in the 1913 edition.[7]

Thalbitzer has put forward claims for lesions in Helweg's triangle in the spinal cord in this disease.[8] A superficial review of available Danvers material by R. L. Van Wart of New Orleans, Louisiana, failed to show more alterations in available manic-depressive cases than in other conditions. A careful study of this region in many conditions is demanded; it is doubtful whether light will be thrown on manic-depressive insanity thereby.

The somewhat surprising results of a recent analysis of our Danvers cases of dementia præcox, viz., 86 per cent of the cases demonstrating macroscopic or microscopic lesions, often of a suggestively focal character, gave point to a re-investigation of manic-depressive material from the same source. The presence or absence of similar lesions, studied especially from the topographic point of view, would obviously offer one of the best possible controls for the dementia præcox work.

The presence of focal atrophies, aplasias, or scleroses in silent areas of the brain, *without appropriate symptoms or without any symptoms,* as I point out more fully elsewhere, would lead us to the old Meynert conception of "functionally unoccupied" areas, not yet filled or otherwise utilized by experience. This conception of Meynert's has been largely replaced to-day by the notion that these so-called silent areas really have a message for the right receiver. The pathologist feels bound to explain, as best he may, in some functional terms any "brain spots" he may discover. The "normal" brains of the general hospitals, prone as they are to show now and then massive cut-outs, such as cysts of softening, fail to show, as I shall shortly bring out by a detailed analysis now in preparation, the kind of thing I have described for dementia præcox.

But grant, for the moment, that such things as normal brains exist and come to autopsy, how stand manic-depressive brains? Do they stand with the normal brains or with the dementia-præcox brains?

A word concerning the intimate nature of the lesions in question: At the first symposium of the New England Society of Psychiatry dealing with manic-depressive insanity, I reported the surprising paucity of brain material "normal" *in all ways* in the series then available. Only one of 37 brains seemed normal in all macroscopic and microscopic respects. One critic jumped to the conclusion that the abnormalities discovered, various as they were, running all the way from sculptural anomalies to general cytopathological changes, were assumed by me to be correlated with the disease. Such was, of course, not my intention, but merely to indicate how difficult is this region of analysis. Evidence of actual destruction of brain tissue, eventuating as a rule in macroscopically recognizable lesions, is the species of

evidence of the greatest value at this juncture. I am far from discounting the scientific value of cytopathological changes *per se* or denying possible importance of, *e. g.*, neurofibrillar changes.

You are all aware, also, of the bold claim, recently made by Alzheimer,[9] that cases of catatonic excitement can be told from cases of maniacal excitement on the score of certain disintegration products present in and between the cells of the catatonic brain. Similar disintegration products, Alzheimer states, may be found in the brains of cases showing severe visceral disease. Here lodges a tremendous difficulty; for precisely manic-depressive patients, dying in attacks, are often subject to severe visceral, apparently non-nervous, disease. To resolve that difficulty will require, I believe, the most careful inquiry into the *distribution* of these products, as well as of other cytopathological changes, in the different brain-parts, and much keen clinical correlation.

It is true, too, that even such comparatively unequivocal evidence of nerve-cell injury as the development of satellitosis is often made to bear too heavy a burden of explanation. It was a very salutary thing that, at the second symposium of the New England Society of Psychiatry on manic-depressive insanity, S. T. Orton, of Worcester State Hospital, Mass., showed much evidence of satellitosis in manic-depressive material. Here, again, the important question, from the entity's standpoint, is the constancy, degree, and habitual regions of these destructive changes.

An abstract of Orton's conclusions,[15] based on his entirely independent data, studied without reference to my own work, is as follows:

ORTON'S WORK ON SATELLITOSIS IN THE PSYCHOSES.

The analysis of the relative numerical occurrence of satellite cells in 10 cases in each of five psychoses seems to warrant the conclusion that satellitosis cannot be considered in any sense indicative of the type of psychoses, although it has in this series appeared with more consistent intensity in the manic-depressive cases and has been of very much less prominence in dementia præcox.

The reaction elects the deeper cell layers both in regard to frequence of occurrence and degree of reaction.

The cortices of the dome, precentral, postcentral and frontal, seem to show the reaction with greater intensity than do the temporal and occipital regions.

Age at the time of death seems to play some part in the occurrence of severe reactions, but cannot be considered the only factor.

The duration of the psychosis bears no demonstrable relation to satellitosis.

It is not the mere presence, then, *of some kind of destructive changes somewhere in a brain* which promises to solve these most difficult clinical problems. Decades of experience with *état criblé* and cortical arteriosclerosis in general hospitals should alone dispose of so crude a hypothesis.

The first question of our study is this: *Do the brains of manic-depressive cases,* studied by the same methods employed in work on dementia præcox, *show anomalies or scleroses similar to those found in dementia præcox?*

II. Analysis of Case-Groups, with Special Reference to Excluded Cases.

Available for an orienting study were 49 cases of mental disease in which the diagnosis of manic-depressive insanity had been made on criteria, largely Kraepelinian, at Danvers Hospital. If some of these were really cases of dementia præcox, they might possibly stand out as such on the basis of lesions which they would prove to have. From prior consideration it seemed best to exclude 6 cases of involution-melancholia (811, 821, 895, 1397, 1399, 1419), which happened to be all female. We remain with 43 cases, numerically a proper foil to the 37 cases of dementia præcox supposedly "non-organic" which we before studied (1910).

Of these 43 cases, the following presented gross lesions of interest in this connection: 1067, 1097, 1156, 1170, 1173, 1277, 1284, 1305, 1327, 1356, 1373, 1426; *i. e.,* 12 cases, or 28 per cent. These lesions include scleroses, atrophies and anomalies, wherever found, but do *not* include hemorrhages, cysts of softening, or other focal lesions, which occurred in 4 other cases. Should we exclude these 4 cases from our 43, we obtain as a percentage of manic-depressive cases having focal convolutional scleroses, atrophies, or anomalies (12 in 39), 31 per cent.

The above-detailed cases, it will be remembered, formed a group of 12 with lesions and anomalies more or less similar to those found in the dementia præcox study (1910).

1067 should be at once excluded both from numerator and denominator, leaving 11 in 38.

NOTES OF THREE CASES REGARDED AS DEMENTIA PRÆCOX.

On the whole, I should myself be tempted to consider 1097, 1305, and 1373 as more fitly placed in the dementia-præcox group, and should exclude these also from both parts of the fraction, leaving 8 focal lesion cases in 35. On account of the importance of *not* excluding any focal-lesion cases of manic-depressive cases by a *post facto* diagnosis of dementia præcox, I present condensed histories in these three cases.

G. M., male, D. S. H. 12827, Path. Lab. 1097.

The diagnosis of manic-depressive insanity, depressed phase, was made in this laborer of 29 years, largely on his general appearance and apparent depression. It is doubtful whether there was at any time in the single attack, which lasted in all less than one year from onset, any differential sign of manic-depressive insanity.

The hereditary taint was strong: brother insane, dead at Worcester State Asylum at 29 years; a second brother under treatment for nervous prostration; father alcoholic and a suicide at 50 years; father's brother insane, probably with dementia præcox; mother, one brother and a sister not insane so far as known.

Patient left school at third grammar grade at 14, sustained a fall upon the head about that time, underwent "typhoid-pneumonia" in the Spanish-American War, used little tobacco, some alcohol, had several attacks of gonorrhea and was unmarried. Tried to commit suicide with creolin on a drinking bout at 27 years.

At onset (29 years), morose, loafing about house. Four weeks later stopped work and vanished, only to return with bundles of meat and a complaint of having been robbed of his money. Patient thought the family infested with vermin and combed a baby's hair for hours on that hypothesis.

Signs of pulmonary tuberculosis on admission to D. S. H. December 2, 1905. Tremors general, particularly of tongue and of extended fingers. Idea of vermin constant, possibly based on tactile hallucinations. Vermin also seen. Auditory hallucinations possible. A peculiar "sick" smell at times. Consciousness clear. Orientation for time and persons imperfect (indifference). Vague and shifting delusions (lice, poison, "Place will burn"). Refusal of food, weeping, wringing of hands, appearance of depression.

May 24, 1906, about a dozen generalized convulsions. The lips twitch; eyelids tighten; mouth pulls to left; left arm, then left leg, stiffens; toes of both feet strongly flexed; mouth pulls to right; right eye shifts to

right. Skin livid, breathing stertorous, groaning. Later, arms keep twitching; left side of face finely tremulous; gasping; grimacing, incontinence of urine. Pupils dilated, non-reactive to light. Upward bend of all toes on plantar stimulation. Convulsions suspended by hyoscin.

During the winter many boils and re-infections. Death after increasing weakness and ten days' symptoms of tuberculous enteritis, September 18, 1906.

A review of the case on clinical grounds alone would suggest a preferred diagnosis of dementia præcox.

Anatomical Diagnosis.

General muscular atrophy.

Marked malnutrition.

Sacral and trochanteric and left iliac decubitus.

Tuberculosis, with cavitation of both upper lobes and right middle lobe of lungs.

Infarct of left upper lobe (occlusion of vessel by caseous material).

Tuberculous ulcers of jejunum and ileum, with peritoneal tubercles.

Tuberculosis of mesenteric lymph-nodes and peribronchial nodes.

Typhlitis and colitis.

Chronic adhesive pleuritis of both upper lobes, right middle lobe and posterior part of right lower lobe.

Chronic fibrous pericarditis.

Chronic fibrous endocarditis.

Slight mitral and aortic valvular sclerosis.

Slight coronary arteriosclerosis.

Chronic splenitis.

Chronic perisplenitis, with adhesions.

Chronic orchitis, right.

Slight aortic sclerosis.

Calvarium dense and heavy.

Chronic external adhesive pachymeningitis.

Chronic fibrous leptomeningitis (right precentral, left superior frontal, right third temporal, sulci, and about pineal body).

Slight sulcal anomalies.

Hypoplasia of left transverse sulcus of orbital region.

Left gyrus rectus narrower than right.

Prefrontal gliosis, especially left.

Cerebellar gliosis (?).

Chronic inflammation of inferior halves of drums, especially right.

No gross lesions of spinal cord.

Head Findings.

Pia mater, in the main, normal; thin and transparent, but thickened in a few foci, particularly about the arachnoidal villi, and especially in right precentral sulcus, in left superior frontal sulcus 4 cm. from frontal pole,

and in the right third temporal sulcus in a plane with uncus. The thickenings in the left frontal and right temporal regions amount to small frank scars, 4 mm. thick in the middle. The tissue of the velum interpositum also unusually dense about the pineal body; there is no especial thickening of the pia about the cisternæ; at the base, the vessels of the cranium show no gross signs of sclerosis. Pia mater strips readily in all parts.

Brain weight 1115 grams.

Fissuration shows trifling anomalies on the two sides; the right transverse sulcus of the orbital region is well developed, the left almost absent. The substance shows a faintly darker color than usual. The left gyrus rectus is narrower than the right. Consistence slightly reduced except in the two prefrontal regions, of which the left is firmer. The hippocampal gyri are not unduly firm.

On section, the left prefrontal region, the sulcal surfaces beneath the scar in left first frontal sulcus, and the external face of the left gyrus rectus are visibly atrophic, being narrower and of a lighter color than the adjacent cortex. The tissue beneath the scar in the right temporal sulcus is not visibly atrophic.

Basal ganglia show no gross signs of lesion.

Cerebellum of uniform consistence, which is slightly subnormal. The laminæ look somewhat narrower than usual.

Olives and dentate nuclei of even and slightly reduced consistence.

A. L., female, D. S. H. 8559, Path. Lab. 1305.

Committed June 9, 1882, as suicidal and vagrant and classed under "chronic melancholia." Father died from some form of "paralysis." An aunt and cousin insane. Delusions of self-reproach and of persecution.

Attacks said to have occurred at 35, at 45 (four months' duration), at 47 (five months' duration). Discharged October 24, 1882. Recommitted March 30, 1893; discharged May 27, 1896; recommitted January 30, 1897. Despite these apparently separate attacks, the whole case presented the appearance of a long-standing dementia præcox of paranoidal trend, with a certain variability of attitude. At times patient's delusions would retire into the background, and her attitude could be characterized as one of reticence on certain topics. The total picture showed now a quiet depression or a surly unresponsiveness or restlessness and quarrelsomeness, or suicidal tendencies. March, 1902, an epileptiform seizure. 1905, amnesia prominent. 1906, increasing feebleness, but maintenance of flesh. 1908, disturbed, complaining, making suicidal threats, disoriented, amnestic, subject to involuntary urination. February 22, 1909, death after three days' acute illness.

Anatomical Diagnosis.

Bronchopneumonia.
Acute bronchitis.
Peribronchial lymph-nodes enlarged.

Injection of intestine.
Congestion of ileum.
Injection of trigone in bladder.
Arteriosclerosis, basilar and of finer branches.
Extreme calcification and atheromatous ulceration of aorta.
Heart hypertrophied, weight 415 grams.
Chronic valvular sclerosis, mitral, tricuspid, aortic, with calcification.
Hydropericardium.
Sclerosis of ventricular walls.
Cloudy swelling heart muscle.
Slight beginning cirrhosis of liver.
Chronic interstitial nephritis, with cysts.
Anomalous position of left adrenal.
Chronic gastritis, with dilated stomach.
Chronic perisplenitis.
Calcification of terminal bronchi (?) in lungs.
Chronic endocervicitis.
Chronic cervicitis.
Serous cysts of Fallopian tubes.
Atrophy of ovaries.
Eversion of left leg.
Edema of lower eyelids.
Unequal pupils.
Teeth absent.
Calvarium dense.
Chronic pachymeningitis.
Slight chronic leptomeningitis.
Cerebral softening, right precentral gyrus and right basal ganglia.
Narrow superior temporal gyri.
Hypoplasia (?) of left superior temporal gyrus.

Head Findings.

Brain weight 1220 grams.

Left hemisphere a trifle longer than right, which presents a blunted tip. Both frontal poles firm; otherwise the left side shows normal consistence, with exception of first temporal gyrus, which is narrow and of lessened consistency as compared with other gyri. In the parietal portion of the right cerebrum, the consistency is much below normal; and the precentral gyrus, 2 cm. from median line, has a softened area 1 cm. in diameter, with loss of contour of gyrus. First temporal gyrus same as on left side. The right caudate nucleus shows a softened area with entire loss of internal capsule, the softened area being about 2 cm. in diameter.

No other areas of softening found.

Cord shows no gross lesion.

J. M., male, D. S. H. 15368, Path. Lab. 1373.

An English saw-maker. Father a suicide. Gonorrhea at 24, with attacks of inflammatory rheumatism at 24 and at 30. Since then attacks

of sciatica, lumbago, muscular rheumatism. Headaches began at 33 and lasted till three years before commitment, when a feeling of pressure at top of head replaced headaches. Wife had one miscarriage. Alcohol and tobacco moderate. Regular churchgoer. "Nervous prostration" at 47, depression, insomnia, restlessness, delusions of self-reproach, spells of praying, elated periods, visual hallucinations, "shaking in the bowels." On commitment, December 6, 1909, orientation good, memory remarkably good, attention hard to secure, auditory hallucinations, persistent belief that "everyone has three doubles," untidiness, motor restlessness, flighty conversation. Death from dysentery, January 6, 1910.

Anatomical Diagnosis.

Bronchopneumonia.
Acute ulcerative colitis.
Mesenteric lymphnoditis, bronchial, retroperitoneal.
Superficial abrasions.
Coronary sclerosis.
Slight basilar sclerosis.
Ventricular endocarditis.
Chronic fatty myocarditis.
Slight hypertrophy of heart.
Fatty liver.
Focal congestion (?) of liver.
Chronic interstitial nephritis, with congestion.
Hypertrophy of prostate.
Distention of bladder.
Chronic perisplenitis.
Scar at apex of left lung.
Thickening of mesentery.
Hypernephroma of adrenal.
Emaciation.
Perforation of left and opacity of both ear-drums.
Slight chronic internal adhesive pachymeningitis.
Slight chronic leptomeningitis.
Encephalomalacia (encephalitis ?) of temporal lobes.
Superior parietal hypoplasia or atrophy.
Frontal sclerosis (*crowns of gyri*).

Head Findings.

Brain weight 1450 grams. *Pons and cerebellum* weight 170 grams.

Convolutions well rounded with exception of *superior parietal gyri* on either side. *These are smaller than normal and depressed* below the surface level. Brain firm throughout, resilient over superior aspect. Cornua ammonis a trifle softer than usual. *Ventricles* smooth. Consistence of temporal lobes softer than any other portion of brain. Choroid plexus slightly cystic. The *frontal regions on section retract from under the*

knife, and the grey matter over the crowns of the gyri is china-white. White matter throughout the brain shows many small bleeding points.

Cord shows injection of pial vessels; otherwise grossly negative.

The outstanding eight possibly manic-depressive cases are:

1156: Probably high-grade imbecile, apparently never schizophrenic.

1170: Perhaps involution-melancholia, apparently never schizophrenic.

1173: Attacks, hypochondriasis, apparently never schizophrenic.

1277: Hysterical reactions, perhaps schizophrenic.

1284: Obscure, delusional, apparently never schizophrenic.

1327: Attacks, depressive, apparently never schizophrenic.

1356: Attacks, depressive, apparently never schizophrenic.

1426: Hallucinations, apparently never schizophrenic.

If we exclude 1170 as involution-melancholia, leaving 7 in 34, we arrive at a group of cases of which only one (1277) yielded phenomena simulating, if not demonstrating, schizophrenia.

NOTES OF TWO CASES EXCLUDED FROM THE MANIC-DEPRESSIVE GROUP.

W. S., male, D. S. H. 13591, Path. Lab. 1170.

Patient was a shoemaker, somewhat given to alcohol, a widower, 64 years. A sister committed suicide at 56, another sister and a brother nervous. Mother nervous, but died at 91. Patient grew tired easily the winter of 1906-7 and began to worry over a strike. Working for a new company, he felt he was being thought a scab and finally stopped work, attempted to choke himself to death with a rope, and entertained delusions of poisoning.

On commitment, April 11, 1907, the main features were delusions of poisoning, anxious depression, hallucinations of taste, smell, and hearing. Death occurred May 12, 1907, as a result of cellulitis of the arm.

Whatever the nature of this case, it does not appear to be a classical instance of manic-depressive insanity. Possibly it belongs to the melancholia group.

Anatomical Diagnosis.

Infection of right arm.

Acute nephritis.

Hypostatic pneumonia, with acute fibrinous pleuritis of posterior third of both lower lobes.

Abscess of muscles of left first intercostal space.

Ecchymoses of scalp and of subcutaneous tissue of abdomen and around prostate and neck of bladder.

Fatty myocarditis.

Coronary, basal, and internal carotid sclerosis (a few foci).

Fibrosis of apices of both lungs.

Chronic adhesive pleuritis of right apex.

Cervical and thoracic myelomalacia (autolytic process hastened post mortem?).

Atrophy of both central regions best marked in *post central gyri,* and especially in the upper third of the left postcentral gyrus.

Erosions of inner table of frontal bone.

Chronic external adhesive pachymeningitis.

Chronic fibrous, leptomeningitis of vertex of cerebello-medullary cisternæ

General encephalomalacia (autolytic?).

Gliosis of lumbar spinal cord.

Head Findings.

Brain weight 1415 grams.

Brain substance shows little variety of consistence and is almost uniformly softer than normal. The hippocampal gyri maintain their firmness to some extent. Upon stripping the pia mater, the convolutions show considerable visible atrophy (or hypoplasia) in the central regions (especially of the left side), but no difference in consistence can be detected with the finger between these convolutions and the rest of the brain. The convolutions of the left central region show the maximal atrophy (or hypoplasia) seen in this subject. The *sulci* appear somewhat abnormally distributed. The upper third of the left postcentral gyrus is reduced to a slender ridge, nowhere over 1 cm. in thickness and tapering somewhat sharply toward the crown. Right postcentral gyrus is also narrower than right precentral gyrus. Section of the central regions of both sides show that the white matter of the postcentral gyri retracts a trifle more from the surface of section than that of the precentral gyri. No similar alterations can be seen elsewhere in the brain.

Weight of *cerebellum, pons and bulb,* 175 grams. Tissue in no way remarkable except for general reduction of consistence.

Ventricles not remarkable. *Basal ganglia* normal. The cervical and thoracic regions of the *spinal cord* show a considerable reduction in consistence, with herniation of white substance from the surface section. The lumbar region, on the contrary, cuts firmly.

L. W., female, D. S. H. 14469, Path. Lab. 1277.

Patient died in the second attack of what may very well be manic-depressive insanity. The first attack was one of excitement at 45, two

years after the menopause, occasioned apparently by reaction to a hoax played by her nephew (elaborate pretense of suicide). The second attack was at 58, regarded as a reaction to a quarrel with certain co-tenants over house matters. The interval was quite clear.

Patient was youngest of five children. An older sister was peculiar and given to violent outbursts. The other three children lived to middle age without insanity. The patient's father was normal and temperate; death at 87. The patient's mother was normal till 85, when senile dementia set in; death at 86. The maternal grandmother was a notorious crank, thought to be insane.

Patient was rather a delicate child, grew robust at 16, was subject to fits of bad temper and was a good scholar; illegitimate child at 23. After marriage, several pregnancies, but only one survival to term, with death of child soon after.

The first attack at 45 showed excitement and depression, with delusions of poisoning and two attempts at suicide. The diagnosis of acute mania was made at one time. Numerous details are available of both attacks; the second attack resembled the first, with auditory hallucinations added. On physical examination, August 11, 1908, there was some question of hysterical anesthesia or of a hysteroid reaction (patient lying flaccid in uncomfortable postures and not responding to pin-pricks, eyelids held tightly together). At first tube-fed, restless and untidy, patient later brightened somewhat and became well-oriented. Hypochondriacal ideas ("numb and paralyzed all over," "cancer of mouth"). Stupor came on once more with same passive but less resistive attitude as before (eyelids kept closed), albuminuria, rapid, irregular heart, puffy face and ankles. Death September 20, 1908.

Anatomical Diagnosis.

Acute diffuse nephritis.
Chronic interstitial nephritis.
Acute inflammation of ileocecal valve.
Acute endometritis, with polypi.
Atheromatous ulcers of aorta.
Slight aortic-valve sclerosis.
Sclerosis of ventricular wall.
Fatty myocarditis.
Edema
Obesity.
Fat-replacement of pancreas.
Fatty liver—passive congestion of liver.
Cholecystitis.
Focal adhesive pleuritis.
Chronic bilateral hydrosalpinx.
Contused wound of nose.
Dilated pupils.

Calvarium dense.

Compensatory subpial edema.

Cerebral atrophy, frontal and precentral.

Head Findings.

Brain weight 1205 grams.

In the *frontal and precentral* regions of both hemispheres there is marked *narrowing of convolutions* and yawning of sulci. Posterior to Rolando and inferior to Sylvius, the convolutions are of about normal width. The vessels of the circle of Willis are soft, and free of demonstrable thickening except at the commencement of left posterior cerebral, at which point there is a minute yellowish plaque. On palpation the hemispheres are of uniform firmness. Ventricles contain a small amount of clear fluid. Ependyma smooth. Cut surface of brain not remarkable. *Basal ganglia* normal. Weight of *cerebellum and pons,* 155 grams; not remarkable.

Cord not remarkable.

If one followed the Bleuler concept of dementia præcox as schizophrenia, there would thus remain 6 cases showing focal lesions or anomalies in a group of 33 cases of manic-depressive insanity. Although I acknowledge that the diagnoses in these 6 are not all trustworthy, yet we shall be overstating rather than understating the percentage of focal brain appearances in manic-depressive insanity if we state it on this basis, namely, 18 per cent.

Here is the group:

1156, 12004: Female, onset at 19, attacks, death at 25; regarded as manic-depressive, but possibly as dementia præcox and probably as in any case somewhat feeble-minded.

1173, 13461: Female, onset at 28, attacks (perhaps not well in intervals), death at 62; "hypochondriasis on a psychasthenic basis, possibly manic-depressive" (son a Danvers patient).

1284, 14583: Male, onset at about 50, death 17 days from onset; intensely hallucinated, hyperreligious, self-accusatory, following exile from Turkey (Armenian) and loss of property in Chelsea fire. The diagnosis must remain in doubt.

1327, 15061: Female, onset at 50 (8 months), second attack at 73 (death after 5 weeks); strong hereditary taint, probably manic-depressive.

1356, 15251: Male, onset at 60 (suicidal, 6 months), second attack at 65 (death after 7 weeks); very strong hereditary taint, probably manic-depressive.

1426, 15724: Female, onset at 52, death after 5 or 6 months of symptoms, possibly manic-depressive.

I assume that 1156, 1173, 1327, and 1356 might be generally accepted as manic-depressive cases; and removing 1284 and 1426 as too fulminant for diagnosis, we should arrive at 4 focal cases in 31, or at least 13 per cent.

EXCLUDED AS TOO FULMINANT FOR ACCURATE DIAGNOSIS.

K. M., male, D. S. H. 14583, Path. Lab. 1284.

Armenian, about 50 years old (grandfather a suicide), thought to have brooded over his exile from Turkey, overworked and exhausted. Lost furniture in the Chelsea fire. Two days before commitment, refused to go to work in shoe factory, confessed his sins to the priest, and told his son to pray. Visual hallucinations. Ideas of self-reproach (cause of the Chelsea fire). Persistent beating his head and eyes. On commitment, October 3, 1908, beat his eyes and eventually blinded himself. Disorientation complete. Restlessness. Persistent talking about sins, Turks and the government. Stuffed ears with rags (auditory hallucinations?). Death October 18, 1908.

Anatomical Diagnosis.

Partial absorption and suppuration of both eyeballs.
Extensive abrasions of face, neck and chest.
Abrasions of both olecranons and of both feet.
Acute nephritis.
Hypostatic pneumonia of right side.
Acute fibrinous pleuritis.
Edema and congestion of base of left lung.
Enlargement of bronchial lymph-nodes of right side.
Acute splenitis.
Congestion of pia mater.
Enlargement of duodenal lymph-node.
Chronic ventricular endocarditis.
Sclerosis of mitral valve, aortic arch, coronary and basilar arteries.
Fibrosis of border of liver.
Slight cirrhosis of liver.
Emaciation.
Arachnoidal villi in excess.
Prefrontal atrophy.
Prefrontal, frontal, and occipital gliosis.
Cerebellum and cord soft.

Head Findings.

Brain weight 1385 grams.

The anterior and posterior poles are firmer than the central and ventral regions of the brain. The cortex, on section, is a trifle greyer in these

regions than elsewhere. The prefrontal region shows a slight narrowing of cortex. Gyri in general of a normal richness and appearance. The puncta cruenta of the interior do not seem unduly injected. No granular ependymitis was demonstrated.

Cerebellum, weight with *pons,* 155 grams, soft.

Spinal cord not remarkable.

D. P., female, D. S. H. 15724, Path. Lab. 1426.

Normal until 52 years, when influenza kept patient in bed a week and left her weak, depressed, "nervous," delusive about neighbors, crying in spells. Later, auditory hallucinations, explained as delusions. Eventually, ideas of self-reproach and, four months after onset, attempt at suicide with razor. After recovery from this attempt, self-reproachful ideas persisted and deepened. Death after 5 days of dysentery, 26 days after commitment.

Anatomical Diagnosis.

Acute hemorrhagic colitis.
Localized fibrinous exudate over colon.
Beginning bronchopneumonia.
Congestion of lungs.
Cloudy swelling of heart muscle.
Atrophy of spleen.
Chronic inflammation left internal laryngeal wall.
Chronic interstitial thyroiditis.
Scar of neck.
Sclerosis of coronary arteries, aorta, left carotid and innominate arteries.
Edema in legs.
Anemic thoracic muscles.
Chronic interstitial nephritis, with cysts.
Chronic adhesive pleuritis.
Scar at apex.
Chronic focal perihepatitis.
Cystic glands of cervix uteri.
Cystic organ of Rosenmuller.
Injection of Fallopian tubes.
Slight splanchnoptosis.
Asymmetry of face.
Lüeniæ atrophicæ.
Calvarium dense and thick.
Marked pigmentation of pia over medulla.
Atrophy of left third frontal, left precentral and right postcentral gyri.

Head Findings.

Brain weight 1280 grams.

Frontal lobulations plump. Considerable gaping of sulci around left third frontal convolution, with distinct depression at beginning of left Sylvian fissure. Left prefrontal convolutions much narrower than the right while the left postcentral is slightly larger than its fellow on the right. No areas of softening. Slight increase in resilience in the right prefrontal region. The *pia* strips from brain easily.

If we look more narrowly at the four remaining (viz., 1156, 1173, 1327, 1356), having in mind anatomoclinical correlations, we are not astonished at some of the things found. Thus, the fact that 1156, suspected of imbecility, should show small and overnumerous convolutions in the left superior and middle frontal region (as well as microscopic changes) is not surprising; but the findings may have little or nothing to do with the manic-depressive phenomena.

1173, again, shows frontal lesions, in the form of a bilateral atrophy; there was also a somewhat generalized gliosis as indicated by induration, involving both cerebrum (especially occipital regions) and cerebellum. There were various acute changes (including axonal reactions) in all parts of the central nervous system examined (death ascribed to chronic Bright's disease). The correlation between the frontal emphasis of the lesion and the generally delusional nature of the symptoms is striking. Otherwise the whole nervous system may be said to have reacted with equal mildness to the degenerative process, whatever it was.

1327 was another instance of frontal emphasis of lesions, here confined to the two prefrontal regions. The brain weight indicates a loss of perhaps 100 to 110 grams, with some internal hydrocephalus. There was also in this case a marked cerebral arteriosclerosis, together with an internal capsular cyst of some standing.

1356 showed also a generalized mild induration of brain and cord, with a tendency to atrophy or aplasia of the left postcentral and right superior parietal regions. This case had considerable disorder of consciousness from time to time, terminating in periods of depression with delusions, and consequently regarded as due thereto. Perhaps the case belongs in one of Kraepelin's newer subgroups in dementia præcox (1913).

I give, below, these:

FOUR CASES, WITH FOCAL BRAIN LESIONS, REGARDED AS POSSIBLY BELONGING IN THE MANIC-DEPRESSIVE GROUP.

K. T., female, D. S. H. 12004, Path. Lab. 1156. This case was termed one of manic-depressive insanity, depressed phase, but dementia præcox was a diagnosis also entertained. A "constitutional basis" was maintained. It is possible that patient should be regarded as an imbecile of high grade, with occasional suicidal and depressive attacks.

The patient was an English girl (father intemperate, an uncle insane after stroke, another uncle epileptic) who came to the United States at 8 years, finished school at 14, worked in mills till 19.

August, 1901, patient grew despondent after menses ceased; October 13, cut her throat with a carving-knife; and was committed to Westboro State Hospital, November, 1901. Discharged recovered May 1, 1902. Patient then began to lead an irregular and immoral life, and was committed to Danvers State Hospital August 7, 1902, with insomnia, pains in head, and threats of violence. Patient became euphoric and amiable, but about October 1, 1902, began to be depressed and to have crying spells, and to find fault. Excitement, abusive and profane language, and sauciness followed. Discipline by transfer from one ward to another was usually successful in changing mood. Discharged on trial visit March 25, 1903.

Recommitted June 27, 1904, after spasmodic attempts to go to work and resumption of irregular life (gonorrhea). Discharged much improved on trial visit July 5, 1905, but was returned September 27, 1905, after another resumption of immorality, in a restless, euphoric state. Tubercle bacilli were demonstrated in the sputum in June, 1906. Moods were variable. There were some outbreaks of sharp excitement, other short periods of depression. Phthisis began early in 1907. Sacral bedsore. Ischio-rectal abscess. Death March 24, 1907.

Anatomical Diagnosis.

Tuberculosis of lungs, with cavitation and bronchiectasis.
Tuberculosis of bronchial lymph-nodes.
Tuberculous ulceration of jejunum, ileum and colon.
Enlargement of mesenteric lymph-nodes.
Miliary tubercles of liver.
Miliary tubercles of kidneys.
Amyloid reaction of liver and spleen.
Emaciation and anemia.
Sacral decubitus.
Decubitus of heels.
Aortic sclerosis.
Chronic fibrous myocarditis, especially of left auricle.
Chronic fibrous endocarditis, especially left ventricle and auricle.
Slight mitral sclerosis.

Slight coronary arteriosclerosis.
Serous pericarditis and peritonitis.
Chronic adhesive pleuritis.
Chronic external adhesive pachymeningitis.
Slight chronic leptomeningitis (parietal).
Slight tendency to microgyria of left superior frontal and middle frontal convolutions.
Calcified plaques in lumbar pia mater.

HEAD FINDINGS.

Brain weight 1310 grams.

Consistence is not especially firm. Convolutions over the *first and second frontal* regions on the *left* are *smaller* and *more numerous* than the corresponding area on the right. On section the tissue is bloodless. The ventricles are free from granulations, no sclerosis in the basal vessels.

Spinal cord: Pia over posterior surface of cord has numerous whitish thickenings; otherwise not unusual.

M. B., female, D. S. H. 13461, Path. Lab. 1173.

"Hypochondriasis on a psychasthenic basis." "The diagnosis of manic-depressive insanity can be stretched possibly to cover this case." Always peculiar, patient had possibly nervous prostration at 28 years, after the birth of a second child. (This child, a son, was a patient in D. S. H., 7654). Thereafter always ailing and taking patent medicines. Developed ideas of liver trouble and of slivers in food and on clothes. Committed at 53 to D. S. H., December 3, 1898, and discharged May 1, 1899, much improved. Recommitted January 11, 1907, with delusions concerning liver disease, contamination by dust, and splinters in her clothes, the latter probably based on hallucinations. Death May 24, 1907, with chronic nephritis.

ANATOMICAL DIAGNOSIS.

Interstitial nephritis.
Sclerosis of the aorta, common iliac, and left coronary arteries.
Fibrous endocarditis.
Emphysema of lungs.
Chronic splenitis.
Chronic passive congestion of liver.
Chronic fibrous pleuritis of both sides.
Dilation of the stomach.
Gastroptosis.
Slight emaciation.
Degenerative myositis.
Ecchymoses of the skin.
Tumor of pituitary.
General gliosis of brain, especially occipital.
Frontal atrophy (or hypoplasia?).
Cerebellar gliosis?

Head Findings.

Brain weight 1320 grams.

There is increased consistence of the brain, greatest over the occipital poles, less in the frontal regions, with decreased consistence of the paracentral regions. The frontal convolutions show a narrowing, with widely gaping sulci. On section, the brain substance is firm, and the grey matter shows no atrophy.

Weight of *cerebellum and pons* 145 grams. Cerebellum firm, shows slight increase of consistence.

Small tumor 3 mm. in diameter on anterior surface of pituitary body

Spinal cord: No gross lesions.

M. J., female, D. S. H. 15061, Path. Lab. 1327.

A case with two attacks of mental disease, at 50 and 73 years. Both attacks were depressive, the first regarded as due to prostration attending a severe burn, the second without obvious reason. The first attack lasted eight months and was attended in hospital by a few feeble attempts at suicide, and showed apprehensiveness and depression, with some hypochondriacal and suspicious ideas. The second attack lasted five weeks and showed marked apprehensiveness, mild restlessness, slight emotional depression, ideas of self-reproach. There seemed every prospect of recovery from this attack, as there were no signs of cortical arteriosclerosis and little or no impairment of memory. Death was due to an intercurrent cystitis, with hemorrhages from the bladder wall.

Heredity: Mother twice insane at puerperium. Mother's sister died at 72, a senile dement for 6 years. Patient's only brother alcoholic and only partially self-supporting.

Anatomical Diagnosis.

Acute diphtheritic and hemorrhagic cystitis.
Distention of bladder.
Retroperitoneal lymph-nodes enlarged.
Acute proctitis.
One ulcer in colon.
Acute metritis.
Acute cervicitis.
Hemorrhagic infarction(?) of spleen.
Chronic interstitial nephritis, arteriosclerotic type.
Chronic hepatitis.
Cholecystitis and cholelithiasis.
Chronic interstitial pancreatitis.
Chronic perisplenitis.
Atrophy of spleen.
Sclerosis of aorta, coronaries, and internal and common iliacs.
Slight ventricular endocarditis.
Hypertrophy of heart.

Large amount of epicardial fat.
Atrophy of stomach.
Hemothorax.
Chronic fibrous obliterative pleuritis.
Chronic interstitial fibrosis of lung.
Central softening of adrenals (postmortem?).
Anomalous ureter (right).
Unusual blood-supply of kidney (left).
Mammary glands atrophic.
Teeth poor.
Arrest of development in right hand.
Scars on forearm.
Dislocation of right wrist.
Inequality of length of legs.
Unequal pupils.

Head Findings.

Brain weight 1195 grams. Weight of brain stem and cerebellum 145 grams.

Considerable atrophy and moderate sclerosis of the prefrontal region. Consistence of the remainder of brain about normal, except the temporal convolutions, which are rather soft. On section of brain, an area of white softening, about 1 cm. in its greatest diameter, is found in the anterior portion (also superior) of the left internal capsule. Lateral and third ventricles moderately dilated. No granulations in ependyma. *Cerebellum* not notable. Cerebrospinal fluid increased in amount.

A moderate subdural hemorrhage over the dorsal surface of the *cord* from the third cervical to the second dorsal segment. The right lateral columns of the cord are somewhat lighter in color, in the region of the fifth and sixth cervical segments, than the left. Some softening in sacral region of cord. Surface of section not notable, except that the anterior horn on the right side, in the midcervical region, is more red than the left.

W. B., male, D. S. H. 15251, Path. Lab. 1356.

A physician, with two attacks of depression, the first at 60 years, after several suicidal attempts and lasting about 6 months, the second at 65, terminated by death after seven weeks (dysentery). The second attack (committed to D. S. H. September 24, 1909) showed some ideas of self-reproach and of a developing cancer, so that the case suggested involution melancholia. This phase of the disease was terminated (October 13, 1909) by restlessness and a screaming, trembling outbreak of almost hysterical character. Improvement was then rapid until November 4, when ideas of self-reproach of a sexual character emerged suddenly, followed by confusion, restlessness, anxious expression and general tremors. This condition deepened until no responses could be obtained. Restlessness in bed. A fall, with bruise of hip. Rapid loss of weight.

Some *hereditary data* are available. The maternal grandmother bore to a second husband one daughter, who died at McLean Asylum. Patient's mother became insane after childbirth. Patient's oldest brother (J. B.) blind from "rheumatic iritis"; sister (M. B.) died of myxedema; brother (M. A. B.) a suicide in financial embarrassment; sister (S. B.) gloomy after husband left her, remarried for pique, diabetic; sister unmarried; sister (C. B.) rheumatic, married, mentally normal; brother (A. B.) died of hemorrhage of lungs; brother (W. B.) living, normal.

Anatomical Diagnosis.

Bruises of skin over legs and thorax.
Thrombus in aorta.
Acute nephritis.
Ulcerative duodenitis and proctitis.
Injection left vocal cord and pharynx.
Injection of pituitary.
Icteric conjunctivæ.
Fatty myocarditis.
Hypostatic congestion of lungs.
Healed tuberculosis of right apex.
Aortic sclerosis.
Small spleen.
Atrophy of liver.
Slight fibrous endocarditis and fibrosis of aortic valve and ventricles.
Unequal pupils.
Hypertrophy of prostate.
Skull thin.
Chronic external adhesive pachymeningitis.
Chronic fibrous leptomeningitis.
General cerebral gliosis.
Focal cerebral atrophy.

Head Findings.

Calvarium thin, with moderate amount of diplöe.

Dura mater very adherent, removed with the skull-cap. *Pia mater* irregularly thickened.

Convolutional pattern well preserved. The sulci in the right upper occipital and the left ascending parietal regions are markedly gaping; tissue in immediate vicinity softened. The cortex otherwise seems generally firmer than normal, as does the cord.

Brain weight 1355 grams. *Pons and cerebellum* weight 175 grams.

Basal vessels soft. *Ventricles* smooth. *Pituitary* firm, shows much reddening at extremities.

III. Analysis of Case-Material, with Special Reference to Cases Showing Anomalies, Sclerosis and Atrophies.

Accordingly, at the conclusion of the orienting analysis of all available manic-depressive material in a group of about 500 autopsies, I found myself with a very small number showing lesions and anomalies of the type with which I had become familiar in dementia præcox. The findings may be tabulated as follows:

Manic-depressive diagnosis (clinical)	49
Exclude as clearly involution-melancholia	6
	43
Exclude as complicated by hemorrhages, cysts, etc	4
	39
Exclude as clearly an error of diagnosis	1
	38
Exclude as dementia præcox	3
	35
Exclude as of very doubtful diagnosis	2
	33
Exclude as of somewhat doubtful diagnosis	2
	31

11 of the 38 cases (or 29 per cent) showed lesions of the focal type. 3 of these (on evidence given above) seem to me to belong to the dementia-præcox group. 8 of 35 (or 23 per cent) remain of the focal-lesion group. I think the evidence above given would also go far to warrant the exclusion of two others (see 1170 and 1277) from the manic-depressive group. If we accordingly exclude these, we arrive at six focal-lesion cases in 33 (or 18 per cent), in two of which the diagnosis is surely not possible to establish. A residuum of 4 cases in 31 (or 13 per cent) remains.

Of course, I do not mean that the problems presented by these excluded cases are solved by the mere process of exclusion. Indeed, we are heaping up a vast deal of trouble by so excluding them. But, in an orienting view of the manic-depressive problem, it is necessary to take cognizance of cases which belong to the classically accepted group. It seems to me that the above data

show that far more cases of manic-depressive insanity are free from focal lesions than are cases of dementia præcox examined by the same methods (1910).

It remains to be inquired how far a more careful analysis of a series of brains from the point of view of systematic photography will make or break this provisional hypothesis. We shall enter upon this photographic analysis feeling certain that we shall find no great number of focal-brain cases, but we shall perhaps be less sure of not finding cases of generalized mild atrophy. Inasmuch as certain cases of dementia præcox also show generalized mild atrophy, we should have to fall back on the hope of finding in the comparison of the two groups something differential microscopically. This hope is for the moment slim in view of Orton's study of satellitosis, which (see above) occurred in both dementia præcox and the manic-depressive psychosis.

IV. Notes from the Literature on "Organic" Cases of Manic-Depressive Psychoses.

Several writers have upheld the idea that those cases of manic-depressive insanity which issue in dementia exhibit organic brain changes. Pilcz, in 1900,[11] collected some evidence in this direction both from the literature and from v. Wagner's Vienna clinic. Recognizing possible hereditary factors, Pilcz lays stress on certain acquired factors, and particularly on injuries to the head. Head injuries may work indirectly by providing a *locus minoris resistentiæ* for the hereditary factors, but they may also work directly by providing painful scars which might be conceived to act reflexly, creating mental disorder. Pilcz reminds us of Lasègue's "cerebral cases,"[12] of Krafft-Ebing's concussion cases,[13] and of v. Wagner's claim[14] that brain injury may be of such nature as to produce insanity directly, without recourse to the idea of hereditary taint.

Over and above head injuries, Pilcz became especially interested in *Herde* of other causes, particularly those foci which are of embolic or arteriosclerotic origin, and draws the general conclusion that *dementia in these periodic cases is always attended with focal brain lesions.* "Der Sitz des cerebralen Herdes hat nichts characteristisches." Wollemer's case showed multiple cortical and thalamic foci of sclerosis.[15] Schüle's showed circumscribed

cortical " hyperplasias " and a tumor of the clivus [16] (chordoma?). Kirn's case showed cerebral and midbrain atrophy with cranial asymmetry.[17] Worcester's case showed pontine and quadrigeminal cysts of softening.[18] Savage's case showed scar of frontal lobe, pontine lesions.[19] Charron's first case showed left frontal cyst.[20] Charron's second case showed cysts of softening in right frontal region. Doutrebente's case showed circumscribed unilateral frontal meningoencephalitis.[21] Pilcz's first case showed sclerosis of the right dentate nucleus of cerebellum. Pilcz's second case showed scars of right frontal, left frontal and orbital, left superior temporal, and left temporal pole.

Pilcz therefore found 10 cases with an assortment of focal brain lesions, all in dementing cases. He found eight cases sufficiently described so that he could state that they were anatomically negative. Pilcz then introduces a hypothesis that such *anatomically negative and intellectually intact cases* will probably *show,* upon proper methods of examination, *teratological changes,* such as convolutional anomalies, developmental disorders, factors dependent upon " eine *ab origine* fehlerhafte Anlage des Centralnervensystems."

In brief, Pilcz has reduced the focal lesions to the humbler position of accounting for dementia, and will seek otherwise in the nervous system for signs of the constitutional or hereditary basis of the disease. He believes that certain *hirncongestive Zustände* are possibly accounted for by focal vascular lesions.

Following Pilcz's work, appeared numerous publications dealing with the " organic " idea in periodic mental disease. In 1908, Hoppe published an analysis of 15 cases, autopsied during seven years at the Allenberg provincial asylum of East Prussia (Dr. Dubbers' clinic).[22] Hoppe failed to find the congenital anomalies suspected to exist by Pilcz. Secondly, Hoppe refuses to agree with Pilcz that all dementing cases will be found to have brain scars, but concedes that the signs of chronic brain disease (loss of nerve elements, gliosis, atrophy, hydrocephalus, ependymitis, chronic leptomeningitis) are found in connection with dementia.

On the other hand, Hoppe agrees with Pilcz in his claims that brain scars may serve as irritating factors in the production of mental symptoms of the sort found in the periodic mental diseases. The clinical picture deviates somewhat from that usually found.

The brain-focus cases give more the impression of twilight states such as occur in epilepsy. The excited states are not joyous and not attended by flight of ideas. The depressive phases of these cases may sometimes exhibit emotional dulling, peculiar attitudes, great variation in the clinical picture, and even dementia; in short, these cases may distinctly recall catatonia.

Indeed, the question of catatonia is expressly raised by Hoppe for his thirteenth case, one of pial cyst at the base of the brain, possibly of congenital origin. This cyst occupied the site of the absent right hippocampal gyrus and had pushed to one side the temporal lobe, the pons, and the right cerebellar hemisphere. (A remarkable molding of the temporal lobe had taken place, recalling the conditions of a case published by Ayer from the Danvers laboratory.[23])

A somewhat similar case, in which the suspicion of catatonia has been legitimately raised, was published by Bönhoeffer[24] in 1903 (depression of left parietal bone, catatonic in all general features, but periodic and given to epileptiform twilight states). An operation by Mikulicz, with lifting of bone into place, improved the mental condition, but true epileptic attacks of wide interval came in to supplant the twilight states.

Taubert, 1910, has published an analysis of 42 cases[25] from Siemens' clinic in Lauenburg, Pomerania. Six of these are fully described. Taubert is inclined to separate genetically the dementing factors from those that underlie periodicity in this group.

Among factors which bring out the latent manic-depressive tendencies are cranial trauma, focal brain lesions, and chronic alcoholism. The cases which exhibit no obvious factors, except heredity, have a good prognosis as to dementia. But the dementing cases of manic-depressive insanity, according to Taubert, do not differ markedly from those that do not dement. Like Hoppe, Taubert fails to find the teratological signs in the brains which Pilcz thought would be found (three such cases in Taubert's series were imbeciles). About one-third of Taubert's cases showed wholly normal nervous systems (14 cases=8 normal+6 with simple hyperemia).

V. Special Questions.

An excursion into the literature of the past decade concerning the periodic insanities yields the following problems:

1. What are the respective parts played by heredity, and by focal lesions of the nervous system (trauma and arteriosclerosis)?

2. Are there any evidences of hereditary taint of a visible and tangible sort in the shape of congenital anomalies of the nervous system?

3. Is the dementia which affects some cases of manic-depressive insanity invariably due to arteriosclerotic or other focal lesions?

4. Are the clinical features essentially modified by the incidence of focal lesions of the brain?

The heredity problem admits no clear solution in cases which show, at autopsy, lesions of an obviously acquired nature. To disengage ourselves from an embarrassment of etiological riches, we must take up, if there be such, cases without such acquired lesions. From our own list we have laid aside 6 cases of involution melancholia, whose title to inclusion in the group is *sub judice*,[26 27 28] and 12 cases complicated by focal lesions of a possibly mental-disease-producing character. We remain with 31 cases whose brains, to a rough analysis, are free from disease-bearing factors and are therefore the proper theater for the play of "hereditary instability."

These 31 cases have been thrown into six groups, according to their ages at death. With a few other data, I have set down the main hereditary factors.

GROUP I. DEATH IN THIRD DECADE.

Case.	Sex.	Age.	Duration.	Dementia.	Hereditary factors.
1115 13364	F.	28	14	+	Parents peculiar; father's mother insane.
1353 13069	M.	26	4	0	*Heredity negative.*

GROUP II. DEATH IN FOURTH DECADE.

Case.	Sex.	Age.	Duration.	Dementia.	Hereditary factors.
942 12282	F.	32	5	0	Mother tuberculous.
956 12307	F.	33	1	0	Heredity unknown, post-surgical.
1212 13955	F.	37	5	0	Mother's father hemiplegic; father's father alcoholic; uncles alcoholic, insane; mother insane; sister insane; sister's three children insane.

GROUP III. DEATH IN FIFTH DECADE.

Case.		Sex.	Age.	Duration.	Dementia.	Hereditary factors.
747	7763	F.	41	2	0	Hereditary taint asserted.
774	11131	F.	46	24	0	Father's father insane.
789	11252	F.	46	1	0	Questionable.
959	11657	M.	45	3	0	Negative (data doubtful).
1201	13817	F.	45	32	*	Paternal aunt mildly insane.

* Amnesia.

GROUP IV. DEATH IN SIXTH DECADE.

Case.		Sex.	Age.	Duration.	Dementia.	Hereditary factors.
895	11913	F.	54	13	0	Brother died apoplexy; mother nervous prostration.
899	5840	F.	60	long	+	No data.
926	12250	F.	58	1	0	Brother insane; father tuberculous.
972	11857	F.	56	1	0	Father weak-minded; mother insane.
1005	12421	M.	54	14	0	Father's mother insane; father's cousin insane.
1041	12853	M.	55	20	0	Father tuberculous; mother paralytic; cousins insane.
1238	4457	F.	59	long	+	No data.
1348	14325	M.	57	12	0	Questionable.
1386	15221	F.	53	2	0	*Negative* (but probably involution type).
1425	14108	M.	54	?	0	Mother died of apoplexy; sister epileptic; mother's sister insane, suicide.

GROUP V. DEATH IN SEVENTH DECADE.

Case.		Sex.	Age.	Duration.	Dementia.	Hereditary factors.
761	10004	M.	65	43	0	No data.
821	11394	F.	63	1	0	Brother died after nervous prostration.
864	11558	M.	68	46	0	Both grandfathers drunkards; brother melancholy; two paternal aunts insane; father and sister and daughter easily depressed.
922	9306	M.	65	44	0	No data.
946	12317	F.	63	41	0	Mother demented; two paternal relatives depressed.
968	12040	F.	65	13	..	Questionable.
978	12340	F.	69	7	0	Father's sister insane; brother insane; brother drunkard.
1111	12564	F.	68	18	+	Sister peculiar, probably insane.
1187	13361	F.	62	37	0	Brother insane.
1306	714	F.	66	40	+	No data.

GROUP VI. DEATH IN EIGHTH DECADE.

Case.	Sex.	Age.	Duration.	Dementia.	Hereditary factors.
732 3253	F.	71	18	0	No data.

A further investigation of the cases stated to be *without heredity* yields the following:

Data concerning heredity are absent or extremely meager in 11 cases. The remaining cases yield a surprisingly rich array of hereditary evidence. Only two cases of this group fail to yield such evidence, though these two I am bound to say are pretty convincing.

Thus 80 per cent of the manic-depressive insanities of this group, which do *not* show focal scleroses or anomalies of the nervous system, do give history of insanity in near relatives.

One of the two cases might not be wholly acceptable to a strict critic as manic-depressive, since the case (1386) died in her first attack of depression 30 months after onset at 51 (previous history clear). Perhaps the case belongs rather with the involution melancholias.

The other non-hereditary case deserves more attention. 1353 (13069) was the third of six children, four of whom are well, and the fifth died at 5 years of a spinal injury. Father, Irish, living, always well. Mother, Irish, died at 39. Nothing known of grandparents. No mental or nervous trouble in near relatives.

Patient left school at 14 in seventh grade (failed of promotion once). Obedient, quiet, diffident, not interested in sports. Worked steadily four years in a mill (up to $9.00 per week), later in grocery store for about five years ($8.00 per week), and helped to support family. Smoked freely. Drank but moderately.

Diseases: Measles and mumps as child. Tertian type of malaria at 16, with many chills despite treatment, and recurrence at 17. Grippe at about 19 (apparently a light attack). "Pleurisy" at 21 (sick three to six weeks), but no evidence of this at autopsy.

So far as a pretty adequate history informs us, there were no upsetting factors whatever. April 1, 1906, patient forgot to deliver his orders, became talkative, and grew restless. Patient developed an excessive appetite, complained of indigestion and

incapacity to work, once threatened to cure himself "in the river," and developed insomnia. An exacerbation of excitement led to commitment. The only known factor which could have led to the condition was worry over not securing a new position on a steam railway.

The disease then followed a course of three years and seven months, terminated by bacillary dysentery. Death from bacillary dysentery at 26 years is a rare incident and is, I believe, in our hospital, unique, so that a peculiarly great loss of resistance must be argued in this case. There is evidence of a bilateral otitis media of unknown date, and there was an acute purulent process in one ear after an attack of tonsilitis in January, 1908.

The disease was characterized by numerous short attacks of excitement, distractibility, playfulness (rarely threatening surly attitude), grimacing. Hallucinations were never thoroughly demonstrated, although auditory ones were suspected. There was an occasional suggestion of catatonic mutism and resistiveism; but on investigation these proved to be rather emotional reactions. In the intervals between excitements, patient was a quiet, good hospital worker.

This case would seem to show that *upon no hereditary basis, and without obvious external cause,* a series of maniacal attacks can be produced. In patient's social stratum, perhaps alcoholism in parents and grandparents can scarcely be excluded. Otitis media may have some importance. There was a trifling degree of sclerosis in the lower abdominal aorta.

My conclusion at this point is, therefore, that, whether focal brain lesions produce or essentially modify this disease or not, heredity is a very strong factor statistically (80 to 90 per cent) in a series especially studied. I present one case apparently against any absolute regularity in this respect. It is stringently desirable that cases be reported which fulfil these requirements:

1. Classical or typical manic-depressive insanity.
2. No gross lesions or anomalies of brain.
3. No evidence of heredity (data to be above reproach).

But, if manic-depressive insanity may be a heritable disease, may it also be acquired? The difficulty of resolving this question reminds one of cognate difficulties in the study of epilepsy.

In this direction I have reviewed all the focal-lesion cases, with the surprising result that they practically all belong, to the best of my belief, in other groups of insanity or are remarkably atypical cases. The evidence for this needs presentation in full.

The answer to the question stated above (the existence of brain stigmata) therefore depends upon the attitude you adopt to the diagnoses of manic-depressive made in this group.

The cases with cerebral anomalies, or with lesions which are interpretable as such, are virtually all cases in which you might readily refuse to grant the propriety of the diagnosis.

And, just in so far as you grant the diagnosis to all these atypical cases, in so far are you bound to admit that the lesions do modify the course of the disease. Personally, for the present, I prefer to set these unusual cases aside from the great group of manic-depressive insanity. I should be willing to accord them whatever degree of *alliance* with manic-depressive insanity you please; but this alliance should not, I think, be taken to signify identity.

As to the third question based on the literature (the correlation of dementia with arteriosclerotic or other focal lesions), we deal with 5 cases (or 6, if a case with merely amnesia be included) having dementia amongst the 31. All five were 60 years of age or older at death, except one (1115), who died at 28 after 14 years of symptoms. All had symptoms for long periods. It must be remembered, however, that there were no coarse lesions of arteriosclerotic type in the brains of these cases. The dementia, if arteriosclerotic in origin, must have been due to fine changes not readily observed with the naked eye. In point of fact I regard the hypothesis of an arteriosclerotic origin of dementia in manic-depressive psychosis as entirely arbitrary at the present stage of research. The problem should now be taken up on its merits from the histological point of view, on the basis of clinically unexceptionable cases which have demented—and I venture to think few will be found. I know that many alienists will point to cases with the history of attacks and eventual dementia; but we are beyond the phase of science in which purely clinical evidence is decisive on such a point as this.

As to the fourth question (symptomatology possibly modified by focal lesions), I believe the evidence of the present paper goes

far toward pressing the focal-lesion cases out of the manic-depressive group.

VI. Conclusions.

1. Kraepelin states that the anatomy of manic-depressive subjects is negative. Various authors have described focal lesions with which to account for the occasional dementia which text-books mention. Evidence as to the existence of brain stigmata is equivocal. Orton has recently found satellitosis perhaps rather more in manic-depressive than in dementia-præcox subjects.

2. The fundamental and even practically important question of brain-anatomy in manic-depressive subjects has been here taken up precisely with the same ideas and with similar material as in the writer's first study of dementia-præcox brains—namely, with the topographic idea far more prominent than it has been made by most workers in the field of what used to be called " functional psychoses."

3. The first question which occurs to a critic of my 86 per cent of anomalies, scleroses, and atrophies in dementia præcox is: What percentage of similar conditions would " not-insane " subjects show, and what would be shown in the disease manic-depressive insanity? The present paper deals with the latter inquiry and throws indirect light upon the former.

4. As ever, much depends upon what one terms manic-depressive psychosis. In the text I have given relatively full accounts of most cases excluded from my initial list, which comprised every case which had received the diagnosis (at times on decidedly insufficient grounds) in a certain period at Danvers Hospital. Many of my exclusions tend to swell the dementia-præcox group, and these cases may be studied with my dementia-præcox material of 1910. To avoid confusion I have excluded cases of involution-melancholia.

5. As against my 86 per cent lesions in dementia præcox, I regard 13 per cent as a fair percentage for manic-depressive insanity (4 in 31). A little less rigorous clinical analysis would leave the percentage at 18 per cent (6 focal-lesion cases in 33). In a total random material (after certain obvious exclusions) of 38 cases, it would not be possible, I believe, for the most ardent anatomist to find more than 11 cases of focal lesions (29 per

cent). But this last percentage is assuredly too high, since three cases in the group are pretty clearly cases of dementia præcox. Thus 8 in 35 (23 per cent) is a figure which some analysts might prefer, though personally I believe it too high.

6. Roughly speaking, then, we may think of the *manic-depressive* group as exhibiting *brain stigmata or focal lesions* (*not* arteriosclerotic) in about *one brain in every five,* whereas *dementia præcox* brains show such conditions in about *four out of every five* brains.

7. This finding must be of some significance, whatever the criteria, and whatever particular functional correlations one might infer. The finding does not prove or indicate that the manic-depressive brain is normal; but it does show that the cellular lesions, if any are to be found, must be of a peculiar and probably a reversible nature. And, whereas eager histological researches in the brain are much to the point, perhaps the canny observer will regard the non-nervous organs of the body, or those supplied by the autonomic system, as even more inviting to study in the manic-depressive group.

8. No special histological study is here presented, although some orienting slides have been available in the great majority of cases, from which Orton's conclusions about satellitosis can be in a measure confirmed. Indications of a special line of attack have been presented by Bond in a paper with the writer,[28] and some conclusions bearing on this point have been drawn in the writer's thalamus paper.[29]

9. A study of the literature yielded a few special questions which I have endeavored to answer, largely on the basis of the material without focal lesions, since I regard these four-fifths of my material as far less open to diagnostic suspicion than the one-fifth possessing lesions.

10. The question of the relation of certain instances of eventual dementia to arteriosclerotic brain lesions is provisionally answered in the negative; but the question requires further study.

11. Heredity does not show itself in most manic-depressives in the form of brain stigmata; but the extremely high index of insane heredity in near relatives is remarkable. I am inclined provisionally to regard manic-depressive insanity as constantly or almost constantly hereditary—not in the sense of similar heredity

(this has not been adequately studied), but in the sense that some kind of insanity is almost always, if not always, to be found in near relatives. Without such evidence, I am clinically not now disposed to make the diagnosis "manic-depressive," although it is clear that the rule will not work in the other direction. For the moment, I am challenging my records to produce an unexceptionable case of manic-depressive psychosis which does not show family taint of insanity.

12. Upon these provisional hypotheses, are we to assume that the normal-looking brains of manic-depressives are really normal, *i. e.*, intrinsically,[20] [21] [22] and merely purveying the impulses which a sick body is producing? Or shall we assume a chemical or physicochemical instability of the entire nervous system, such that, although the brain is intrinsically abnormal, the abnormality does not show as yet? Hereditary taint is consistent enough with either assumption, since the germ-plasm might with equal readiness mark the nervous and the non-nervous parts of the body with those invisible marks that produce "functional psychoses."

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XXXVII

STATISTICAL NOTES ON A SERIES OF 6000 WASSERMANN TESTS FOR SYPHILIS PERFORMED IN THE HARVARD NEUROPATHOLOGICAL TESTING LABORATORY, 1913.

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The value of the present series of Wassermann tests is enhanced by the interest which the laboratory officers have taken in the clinical diagnoses of the cases tested and by the courtesy with which the clinicians submitting specimens have given important data.

Since the Harvard Neuropathological Testing Laboratory is not a commercial institution and has performed much charity work in addition to its other work (paid for virtually at cost), a spirit of cooperation has grown up which has helped in the solution of many problems of individual diagnoses.

The Testing Laboratory, of which the last year of work is here summarized, was, I believe, the first to be established in Massachusetts to make use of the ingenious discovery of Wasser-

mann, and, since it has been in continuous action under its various heads, (1) Professor F. P. Gay (now of the University of California) who established the standards since maintained, (2) the late Dr. Emma W. D. Mooers, who brought to the work a great interest in both the technique and the clinical significance of the tests, derived from her work with Plaut in Munich, (3) Professor W. P. Lucas, (now of the University of California) who turned his attention to numerous clinical problems, not only of children, but in general, and (4) their successors (see below), the Laboratory has had a very varied experience and has sustained most of the shocks which the numerous pitfalls of the test provide. The result has been an increasing confidence in the tests as made, and fewer and fewer complaints of lack of congruence between the results of the tests and clinical findings, especially when these latter are reviewed.

My own interest in the tests was in the first instance psychiatric and neuropathological, and I believe that the laboratory would hardly have survived, had it not been for the interest in these tests taken by the state institutions for the insane. The work published heretofore, based on this laboratory's tests, has been largely psychiatric, as the following titles illustrate:

Lucas, W. P. The Wassermann Reaction in its Application to Medicine. Boston Medical and Surgical Journal, clxix, 1913, No. 4, pp. 116-121, July 24, 1913.

Paine, H. L. Results of the Wassermann Test in Two Hundred Consecutive Admissions to the Danvers State Hospital. Boston Medical and Surgical Journal, clxviii, No. 14, pp. 501-503, April 3, 1913.

Morse, M. E. Correlations of Cerebrospinal Fluid Examinations with Psychiatric Diagnoses: A study of 140 Cases. Boston Medical and Surgical Journal (in press) Worcester State Hospital Contributions No. 20, 1914.1.

I believe the value of the Wassermann reaction will always remain even more critical in the neurological arts than in those of the syphilographer who has so many more clinical data immediately accessible to him. It is to be hoped that some state agency will take over the entire task of Wassermann-testing on a scale commensurate with its value. If so, it is the experience of the present Laboratory, which has been virtually supported by cooperation of several of the larger hospitals for the insane, that psychiatric and neurological cases will absorb the lion's share of attention, although from time to time many series of sera will be submitted by physicians having special therapeutic tests (salvarsan, neosalvarsan) in hand.

There are Wassermann tests and Wassermann tests! In comparing our results with others, we often find tests described as Wassermann tests which are somewhat essentially different therefrom. Needless to say some workers omit various precautions and controls which are

essential to reliable work. To make clear the policy of this laboratory, which has been managed in common by the writer, by Professor H. M. Adler, and by Dr. Annie E. Taft under the Department of Diseases of the Nervous System of the Harvard Medical School, I propose to set down (without description) the steps, precautions, and controls used in the series of tests, which are now being executed by Dr. W. A. Hinton.

(A.) *Materials*:

(1) washed sheep corpuscles, 5% suspension, 5 c.c. to tube.

(2) antisheep amboceptor, 2 units.

(3) complement (guinea-pig serum, 10%), 2 units.

(4) patient's serum inactivated at 56,° 0.1 c.c. with each antigen.

(5) antigens (a) human heart reinforced with cholesterin (0.1 c.c.)

(b) human heart reinforced with cholesterin (0.05 c.c.)

(c) alcoholic extract of syphilitic fetal liver (0.25 c.c.)

(B.) *Controls*:

(1) antisheep amboceptor unit tested daily.

(2) Compliment unit tested daily.

(3) 0.2 c.c. inactivated patient's serum used in addition to test with 0.1 c.c.

(4) each specimen tested with three antigens.

(5) control to exclude the (rare) occurrence of natural hemolytic power in complement.

(6) same hemolytic power in amboceptor.

(7) same, salt solution.

(8) control sera six in number (strong positive, slight positive, negative; each antigen doubled in amount.)

(C.) *Remarks*:

(1) period for fixing complements, 1 hour.

(2) doubtful cases repeated with double amount of serum and appropriate controls.

(3) cerebrospinal fluids used in five times the amount of sera.

(4) attention may be called to the "slight positive control (v. B., 8 above) which prevents many slightly positive cases from being overlooked on a given day.

(5) attention is especially called to the use of *three* antigens (This is not usual in many American laboratories), excluding technical error, perhaps indicating slight quantitative changes in a given patient's reaction, and excluding certain false positions.

(6) supervision and control, (highly important where so important a diagnosis as syphilis is being rendered) are obtained by the direct contact of (a) secretary, (b) technician, (c) laboratory boy, in addition to medical expert diagnostician (during 1913, Dr. W. A. Hinton) under general direction of Dr. Annie E. Taft, Custodian, Neuropathological Laboratory, Department of Neuropathology.

GENERAL STATISTICS.

Total Wassermann tests, 1913		6139
Blood sera	5282	
Cerebrospinal Fluids	857	
Total tests repeated		228
Blood sera	186	
Cerebrospinal Fluid	42	
Total individual blood sera		5096
Cerebrospinal Fluids	815	

An important question is always raised by tests which have to be classed as doubtful in result. These doubtful tests occupy a large share of attention and inspire requests for new specimens. I was surprised to learn the low percentage of such doubtful results when all the tests were summed up. The percentage (2-4%) is somewhat like what might be expected of a biological test. The fact that there are more such doubtful tests in sera than in

cerebrospinal fluids signifies doubtless the larger number of confusing substances in the blood as well as other technical difficulties. The figures follow:

Total blood sera	5282
"Doubtful" results (4%)	202
Total cerebrospinal fluid . .	857
"Doubtful" results (2%)	19

It may be properly inquired what is the result of repeated tests in connection with these "doubtful" cases. The following table demonstrates cases in which a change in result was registered involving "doubtful" tests in sera:

"Doubtful" became negative	17
Negatives became "doubtful"	4
"Doubtfuls" became positive	2
Positives became "doubtful"	5

This table would incline one on statistical grounds to prefer to call "doubtfuls" negative. Suppose we look at a group of non-"doubtful" cases to see how they turn with repeated tests.

Negative cases become positive (of which 3 had also been postive also before the initial negative and one had been "doubtful").	15
Positive cases become negative (of which three became positive again later and one had been negative before the initial positive.)	33

If we leave out the anomalous examples of one dissimilar test in a triad, we arrive at

Negative cases become positive 11
Positive cases become negative 29

Comparing this with data above:

"Doubtfuls" become positive 2
"Doubtfuls" become negative 17

we see that, statistically at least, "doubtfuls" resemble negatives more than positives. This also confirms the qualitative suspicions of many workers. Nevertheless it must be pointed out that this general trend toward negative is partly due to natural immunizing or spirochaete-destroying factors at work in the individual and partly to therapeutic efforts.

The figures with respect to "doubtfuls" and changes of reaction in cerebrospinal fluid are as yet too small to make tabulation profitable.

If we exclude "doubtful" tests and limit consideration to the positive and negative tests alone, we have the following denominators:

Blood sera 5096
Cerebrospinal fluids 815

Concerning these blood sera in our laboratory it is not at all certain that they represent the community fairly; indeed it is highly probable that they are a picked class of sera derived from patients in whom syphilis was seriously suspected.

Positive blood sera (23%) 1161

And it is certain that the cerebrospinal fluids are a still more highly selected class.

Positive cerebrospinal fluids
(33%) 267

It is an interesting speculation to consider how far this 33% of positive cerebrospinal fluids represents the chance that an "organic" disease of the nervous system has of being syphilitic.

From the community's point of view it is clear that the percentage of outstanding syphilis is not so high as alarmists have sometimes thought. It is probable from such data as these that the percentage of syphilis in the community is far below 23%.

A curious fact is illustrated by the figures from the Worcester State Asylum, a transfer institution for chronic patients. This institution harbors syphilitics (3 in 103) not quite to the extent of 3% (2.9%.) Here is a community from which the luetic organic cases have largely never been sent. The chances are that this percentage is lower than that of the community at large.

On the other hand, if we consider the girls of the Massachusetts Reformatory for Women, we find the extraordinary percentage of 44, that is, 112 positives in 254 cases.

Some facts concerning the Psychopathic Hospital data were worked up more particularly for the benefit of the White Slave Commission,

and the following paragraphs are reproduced from a report to Dr. Walter E. Fernald, Chairman of that commission.

"I. The general average for the whole state of cases thought by the hospital physicians to be due to syphilis is 5.49% for the three years 1910-1912, and 6.28% for the year 1912 itself. This figure can be safely stated to be far too low because the average percentage of general paresis during the same three years (1910-1912) was 8.43% (for 1912, 8.65%.) This latter figure is also beyond question too small since a certain proportion of coarse brain lesions of epileptic insanities and of imbecilities are undoubtedly due to syphilis, although they are classified outside the syphilis group in the figure presented by the Board of Insanity from which I make the above extracts.

"II. The best information available to me as to the general percentage of syphilis to be found in mental cases is that afforded by our experience at the Psychopathic Hospital covering 1671 separate random tests on different persons. 264 of these 1671 persons were determined to show syphilis so far as the Wassermann reaction carried out under the precepts of the original method can be trusted. I consider that there is no better set of data available for Massachusetts. This will be found to work out a percentage of 14.7% for our

Psychopathic Hospital intake. I think our Psychopathic Hospital intake may be regarded as more representative of the general population than any admissions in previous years to other insane hospitals. An indication of this for example, is the fact that we discharged 250 persons during the last year alone as 'Not Insone' although every one of these cases presented some kind of mental problem. It is rather curious that the percentages which we here work out, viz., approximately 15%, correspond to the idea prevalent I believe among dermatologists as to the general percentage in the community which they deduced from cases resorting to skin clinics.

"III. In a series of random 'organic' cases 321 in number, I found cerebrospinal fluids positive in 118 cases or about 37%. You will of course wonder how I determine cases to be 'organic.' My general policy has been to have examination of the blood serum made in all cases having severe mental symptoms. If the serum is positive, I proceed as a rule to an examination of the cerebrospinal fluid by the Wassermann and other methods, believing it to be of importance to determine whether a case is one of General Paresis or of some other form of cerebrospinal syphilis. In case the serum is negative but the patient shows signs of disorder in the projection system, or as we say, looks 'organic,' then I proceed to a cere-

brospinal fluid examination, despite the negativity of the serum. It is from these two groups that my random organic series of 321 cases is made up.''

Following is not intended as a complete account of the Wassermann technique but only to record the particular materials and main steps used in this laboratory in 1913, so that it may be known exactly what we mean by ''Wassermann'' tests.

SUMMARY.

1. On account of the varying standards and criteria which have held or will in future hold in the matter of Wassermann tests for syphilis, it has been thought wise to summarize the materials, controls, and special precautions used in the Harvard Testing Laboratory.

2. General doubts are often raised as to the reliability of Wassermann's test on account of the ''great number'' of ''doubtful'' reactions; this ''great number'' resolves in our large series to 4% of the blood sera and 2% of the cerebrospinal fluids.

3. On statistical grounds we find the ''doubtfuls'' resolve much more frequently into ''negatives'' than into ''positives.''

4. 23% of all sera examined were positive, and since the cases are in many instances picked as likely to be positive, this percentage is doubtless much higher than the community's total percentage.

5. 33% of all cerebrospinal fluids examined were positive. The principle of selection of these cases was such (positive serum or symptoms of "organic" nervous or mental disease) that the result is of practical value, stateable as follows: The chances of a syphilitic origin for a case of "organic-looking" nervous or mental disease are not more than one in three.

6. The Massachusetts Reformatory for Women yields 44%, a partial index of the infected nature, though not necessarily of the infectivity, of prostitutes and other delinquent women.

7. The Danvers State Hospital (for the insane) yields between 19 and 22% positive sera in its routine intake of cases from Essex County.

8. The Worcester Asylum, a transfer institution (to which are transferred chiefly *non*-paretic cases), yield less than 3% positive. If this percentage should be maintained in future work, one might infer that, from the group of persons in the community with insane tendencies and infected by syphilis, cases are drained off into the frankly paretic group, in such wise that a population of asylum *transfers* will be likely to show a *low* syphilis index. But this conclusion can be only tentative on account of many other issues.

9. The Psychopathic Hospital index (15%) is perhaps somewhat closer to the general community index than the others just mentioned on account of the large number of cases "not

insane'' that are tested, but it is evident that 15% would be too high an index to assign to the syphilis of the general population.

10. Aside from its capacity to solve problems of individual diagnosis, the Wassermann method is obviously of such value to the community that a community Wassermann service might well be undertaken by a state agency such as the Board of Insanity or the Board of Health.

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XXXVIII

ANALYSIS OF RECOVERIES AT THE PSYCHOPATHIC HOSPITAL, BOSTON: I, ONE HUNDRED CASES, 1912-1913, CONSIDERED ESPECIALLY FROM THE STANDPOINT OF NURSING.*

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There is an old Greek saying that we can't be sure of a man's happiness until he dies. And it is certainly true that we can tell much more about most cases of mental disease after their courses are run,—and, if possible, a thorough autopsy has been conscientiously performed, in each case, with scrutiny both of the brain and of the organs of the trunk. Only then can what Kraepelin has termed the longitudinal section of the patient's disease be examined, with all or almost all the body's difficulties revealed or indicated.

So true is this that it sometimes seems to me an impertinence to draw sweeping conclusions from inspection of a brief period of any man's life, especially from the turbid conditions that inform and surround the mental patient's life.

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Particularly perplexing are the cases that recover, whether or no they succumb to further attacks. Particularly, too, those which eventual autopsy determines to have suffered no especial wasting or scarring, or other injury of the brain. Such cases appeared in the Danvers series which I have been working over since 1909 to the number of 235 in 1000. About one case in four, then, failed to yield to the brain examiner any signs of loss of brain tissue, such as would be almost sure to appear had the brain disease been destroying many cells for three months or so before death.

Curiously enough, accordingly, my interest in proving various brains normal (or their conditions in some sense *reversible,* so that the brains, if diseased, should return to normal) has led me to look with especial interest upon the recoveries which the Psychopathic Hospital has more or less to its credit. I use the latter phrase because I had such difficulty in dissuading some of our eager workers from using the term "cure" instead of the term "recovery." Naturally, as in medicine at large, the *vis medicatrix naturae* is often more in evidence than the *vis ipsius medici.*

I have, therefore, concluded to study our recoveries by hundreds until a sufficient basis is obtained for generalizations as to the various factors making for recovery. This first series is somewhat unfavorable for the purpose of generalizations about the major types of recoverable insanity, since, although our problem is only about one-ninth alcoholic, the quick recoveries from various forms of alcoholic mental disease made the alcoholic cases so rapidly outstrip the other recovered cases as to make the alcoholic cases quite dominate the picture.

The following transcripts from charts explanatory of the general scope of our work demonstrate the above statements:

PSYCHOPATHIC HOSPITAL.

Boston, 1913.

For all classes of mental patients *except* regular court commitments.

Of its admissions:

About one-sixth are not insane.

About one-fourth are voluntary.

About one-half avoid expense and "stigma" of court commitment during the year.

1912-3.

Alcoholic Psychoses form one-ninth of

Cases admitted ..... (217-1829)

Alcoholic psychoses 217

Acute hallucinosis 116

Delirium tremens . 64

Other forms 37

1912-3.

All admissions 1523

Remaining in some institution .. 752

Discharged 771

Not recovered .... 340 } 392

Dead ..... 52 }

Not insane ..... 250 } 379

Recovered ..... 129 }

1913.

All admissions 1523

Temporary care 978

Seven days 584

Chap. 395, Acts 1911

Boston police 394

Chap. 307, Acts 1910

Voluntary 362

Other legal forms 183

In partial explanation of these tables and to permit a better understanding of the group of

recoveries about to be listed, let me say that the Psychopathic Hospital is a hospital of 110 beds, which is planned to be operated at about 100 beds. It is a state institution,—an integral part of the Boston State Hospital. It was planned and constructed in such a way as to present itself to the public on an entirely different footing from that of the other state institutions. It is clear from some of the data presented in the above tables that the institution has begun to perform its differential task. For example, out of 1523 patients admitted during the last year, there were 362 which were admitted as *voluntary patients.* Moreover, out of these 1523 patients, there were 250 which we were able to discharge as *not insane.* The "not insane" group was composed not merely of voluntaries, but of various other types of admission. All of these "not insane" cases had had their mental problems to solve. Although these problems are very possibly not in all cases solved forever (many of the patients were of the psychoneurotic group), yet the hospital in so far may be considered to have fulfilled a portion of its new function. It is clear that such percentages of voluntary admissions and of cases which are later discharged as "not insane" are not the rule in existing types of state institutions for the insane, either in this state or in others.

Another feature which differentiates this institution from other state institutions is the very rapidly developing out-patient department, which is now receiving at the rate of over 800 new cases a year. To this department are discharged the recovered cases, and, although the department was not fully organized during the period from which the present series of 100 recoveries is drawn, yet it appears by the records

that 53 of the 100 recoveries appeared at the out-patient department or were investigated by social workers connected with that department.

It may be added that the average stay of patients at the institution is but three or four weeks, although in cases of special diagnostic or therapeutic interest, there is no rule against keeping patients indefinitely, and a number have been kept for relatively long periods for observation and treatment.

With this brief and inadequate sketch of the present status of the Psychopathic Hospital problem, I will pass to a consideration of the first 100 recoveries, referring the interested reader to other official and semi-official publications concerning the Psychopathic Hospital's scope. The first 100 recoveries came within a period dating from the opening of the hospital, June 24, 1912, to about the middle of 1913.

AGE RATIO.

The average age of the group is 40. This demonstrates that our series does not include many cases of the group ordinarily thought recoverable, since many cases with attacks of manic-depressive insanity and of dementia precox should be recovered at far below the age of 40.

The average age of the 30 females was 36; that of the 70 males was 41.

DURATION RATIO.

The average duration of hospital stay in these cases was about 24 days. The duration of hospital stay in the case of women was about four weeks (27 days) on the average; of men, about three weeks (22 days).

The recoveries of the first hundred *persons* involved counting 108 recoveries of *cases,* since there were 7 instances of two recoveries in the same case and 1 instance of 3 recoveries in the same case.

SYPHILIS RATIO.

After routine Wassermann tests for syphilis had been established, 62 serum tests were performed in this group of 100 recovered cases, and of these 62, 9 were positive. The percentage (15%) is exactly the same as that for the total group of 1671 cases performed since our routine was established.

Following are cases in which the condition of the cerebrospinal fluid was of interest, especially in connection with the fact that these are "recovered" cases.

J. D., 1915, 12,010, M., 51, is a case interesting in that he was recovered from what was termed *delirium tremens* despite the fact that he had a positive Wassermann reaction in the spinal fluid, together with a small number of lymphocytes and albumin. The Wassermann reaction in the blood serum was very slightly positive, if at all positive. This may have been due to the alcoholism inasmuch as there appear to be cases in which the alcohol has diminished the Wassermann reaction. The pupils reacted very slightly, if at all, the knee-jerks were diminished and the ankle-jerks absent. There were various areas of anesthesia. Diagnoses of general paresis, delirium tremens, alcoholic hallucinosis and unclassified organic condition were considered. It is possible that this case should not figure as a recovery.

M. H., 11,233, F., 35, also recovered from alcoholic mental disease, regarded as alcoholic hallucinosis (alcohol denied) after twenty-six days' hospital stay, despite the fact that the cerebrospinal fluid gave a

positive Wassermann reaction (serum negative, however). There were some peculiar features in the case which led to a provisional diagnosis of manic phase, manic depressive insanity. Possibly it is a case of syphilitic paranoia.

The patient consistently denied the use of intoxicants nor is there any proof that she drank alcohol. Her one brother is somewhat alcoholic. The illness began with depression and inactivity accounted for by her friends on the basis of the departure of a lover for Ireland. A few days later the patient was dizzy and refused to give her boarder breakfast, stated that she had lost her memory and began to hear bells ringing and people knocking. This phase was rapidly followed by a phase of excitement which was quieted at the hospital by the use of prolonged baths. It was a question whether orientation was wholly preserved on admission.

As to previous physical history, it is stated that patient had had stomach trouble and was troubled with headaches which patient stated were in the top of her head or sometimes in the temples. The physical examination showed the left pupil larger than the right, a slight tremor of lips, a slight systolic murmur at the apex, slightly irregular pulse, moderate edema of ankles. Patient was inclined to be peevish, somewhat resistive and pugnacious, rather slow in speech, sometimes refusing to answer and grimacing. She on this account produced somewhat the impression of being slightly feeble-minded. Mental tests by Binet and other methods demonstrated that patient was from this point of view hardly over eleven years old.

Her pugnacity was easily controllable, her mania was largely at night. She developed a fairly characteristic mania suggesting manic depressive insanity in the first four days after entrance but this rather rapidly subsided in the course of a few days, and patient was discharged on six months' visit, twenty-six days after admission.

Further history is for the time being lacking. The patient was twice introduced into staff meetings for

unclassified cases and the discussion developed suggestions as follows:

Unclassified mania; manic depressive insanity, manic phase; toxic delirium; dementia precox; bacterial infection of the brain; unclassified delirium; acute delirium; infectious psychosis; acute confusional insanity; psychopathic personality, modified by the use of alcohol; mentally deficient with atypical mental state. It was the writer's opinion that the case belonged in the manic depressive group.

As for syphilitic paranoia, or paranoid brain syphilis, Kraepelin in 1910 remarks that the group is very uncertain and almost unknown. Paranoid brain syphilis is a mental disease in which delusion formation and hallucinations are prominent, but physical disorders are not much in evidence. The onset of the disease is sometimes slow and insidious and sometimes tolerably sudden after various indefinite prodromata,—distraught conditions, irritability, excitement, occasionally after an attack of dizziness or a fainting spell. The patients become mistrustful, apprehensive, restless and deluded. Ideas of jealousy are very frequent, sometimes attended with great sexual excitement. Ideas of persecution are especially in evidence, accompanied by numerous auditory hallucinations, as well as others. The patients often have a distinct feeling of being ill; occasionally self-condemnatory ideas are uttered.

As a rule, patients remain quite clear, but there may be transient spells of unclearness. Mood variable, with a tendency to dulling and uncommunicativeness. Kraepelin remarks that it was particularly striking in a series of his cases that there was a sudden occurrence and equally sudden disappearance of extremely violent excitement, with or without external cause.

These excitements were produced by a few words spoken, and immediately after the phase of excitement the patient would become friendly and accessible again, as if nothing had happened. Conduct as a rule quiet and orderly. Seizures are very common. Kraepelin enumerates a series of physical symptoms which occasionally occur and states that in about half of his cases there was a positive Wassermann reaction in the blood. But now and again all these signs were absent, although autopsy later showed the existence of syphilitic cortical disorder. Insomnia and headaches are frequent.

The course of the disease is characteristically very slow. The disorder may retreat completely into the background for long periods. Sometimes there is a condition of more or less pronounced mental weakness, with loss of judgment, senseless delusions, emotional dulling and loss of capacity to work. Kraepelin states that the relation of such phenomena to syphilis cannot be doubted on account of the autopsy findings and calls attention to the fact that similar pictures are observed in tabes.

Kraepelin thinks that most of the cases that belong to this group have previously been placed in the dementia precox or paranoid dementia groups. However, these cases lack the characteristic will disorder of those groups and lack the characteristic cleavage of personality which they would early show. As a rule, patients preserve their outward demeanor well and remain accessible and social. However, in far advanced cases catatonic phenomena are sometimes found.

Several of the patients belonging to this group of Kraepelin were regarded by him for some time as general paretics on account of slight

physical signs pointing in this direction, and on account of the influencibility of their mood and action. However, the absence of speech and writing disorder, the absence of disorientation for time, and absence of all general weakness of the understanding tended to contraindicate the diagnosis of paresis.

DISORDER OF HEAT-REGULATION.

One way of ascertaining how much of a nursing problem these recoveries were is to consider how many cases showed either fever or decidedly low temperature. For assuredly all cases of seriously altered heat-regulation mean good nursing if life and health are to be maintained. Counting cases with 100° or more as showing fever and cases running less than 98° as hypothermic, we find at least 37 cases with either fever or hypothermia. Seven of the 37 had both of these signs at different times. Put another way, there were 23 cases of temperature 100° or above, and 21 with temperature below 98° at some time.

The older writers used to define insanity as chronic non-febrile disease affecting the mind. Whether the disorder of heat-regulation is incidental to the mental disease in the sense of being due to complications, or is in any way more fundamental in some cases, is for the present argument of no consequence. The necessity of good nursing in these cases is clear, and the advantage of general hospital standards in the nurses' attitude to the problem is likewise clear.

In connection with this problem of proper nursing in hospitals like the Psychopathic Hospital, I may be permitted to quote from a letter sent to the committee of superintendents dealing

with the general aspects of the nursing problem for the insane:—

"My opinions are not, I find, as radical as you suggested (doubtless on general grounds) that they would be. I had thought my opinions would be found novel. They are not; since upon my recent trip to Washington, Baltimore and Philadelphia, I found that Dr. W. A. White of the Government Hospital, Drs. Winford Smith and Adolf Meyer of Johns Hopkins Hospital, and Dr. Owen Copp of the Pennsylvania Hospital for the Insane, are in general accord with these opinions. In point of fact, Dr. Winford Smith, superintendent of Johns Hopkins Hospital, is endeavoring to secure exactly the same kind of thing in which I am now interested—viz., the development of a higher type of insane hospital nurse which we may call the psychopathic hospital type.

"I should be in general accord with those who believe in grading, or as developing a stratified nursing force, having at the bottom persons of a custodial type corresponding to the orderlies of general hospitals, and above them a stratum composed chiefly of women of a higher grade. I should of course encourage persons in the orderly or maid servant group to endeavor to rise to the higher or training school grade, but I should be inclined to give up the idea of putting 'round pegs in square holes' by insisting on persons of all grades of intelligence going through the same training school. This can but pull down the general average of the training school.

"As to the training school itself for state hospitals, I should advocate the inclusion of more features recalling those of general hospitals. I should like to have more insistence laid upon the part played by physicians in lecturing and giving practical demonstrations to nurses. In some general hospitals it has been the custom to pay physicians extra for their work in lecturing and demonstrating: this secures better work. In Massachusetts,

however, this plan could not be adopted, but an increase of salary could be granted to persons desirous of spending extra time in this manner.

"Above the two grades just mentioned, I should like to see developed a higher grade of nurses for the insane. The new type of nurse might be termed briefly the psychopathic hospital type. The grade should be founded upon a course pursued subsequent to the general hospital course. The proper length of such a post-graduate nursing course is a matter of doubt but might provisionally be placed at six months. A certificate or diploma should be granted for this work.

"The salary for persons taking this course should naturally be low, perhaps merely enough to cover the cost of uniforms, etc., but the course should be so elaborate, well-conceived and attractive that there would be no difficulty in securing graduates of general hospitals to take the course. Indeed I should say that any course proposed to be of this type which should fail to secure an adequate supply of general hospital graduate nurses would have to be marked down as a failure.

"To develop this grade of nurse, it would be necessary to give the general hospital graduate nurses as good food and living conditions as they have been accustomed to in general hospitals. This would mean placing them in more special quarters and giving them more dignity than is at present accorded to nurses and attendants in most of our state hospitals for the insane.

"So much will suffice for an utterly inadequate sketch of what I shall try to develop at the Psychopathic Hospital in the near future. I do not believe that much money can be saved to an institution which should try to develop this plan, since the money saved upon salaries will undoubtedly have to be spent upon food, quarters and teaching materials.

"I gathered from Dr. White of the Government Hospital that he was of the opinion that such doubly-trained nurses, that is, nurses with general hospital

training plus graduate psychopathic hospital training, would secure good places in private practice. At all events it is clear that these nurses would be better nurses even in general nursing practice than otherwise. But here as in many other proposals, 'the proof of the pudding is in the eating.' To make these general hospital nurses come to us, we must do more for them than in the past and we must not take them from a busy, energetic surgical or medical service to a routine composed of nothing but vigilance and hope to attract a constant stream. One of the troubles with our present system, as general hospital nurses see it, is that they find insane hospital work to consist largely of 'watchful waiting' instead of constructive treatment."

CHANGES IN DIAGNOSIS.

Changes in diagnosis occurred in six cases during a single period of observation:—

11,215 *Alcoholic hallucinosis,* to *delirium tremens.*
11,611 *Delirium tremens* to *alcoholic hallucinosis.*
938 *Alcoholic hallucinosis* to doubtful form of *alcoholic psychosis.*
1,075 *Delirium tremens* to *alcoholic hallucinosis.*
1,774 Same.
1,841 *Alcoholic hallucinosis* to *delirium tremens.*

Changes in diagnosis in persons recovering two or more times, that is, during two or more separate periods of observation which occurred in cases:—

11,007	F.	28	*Unclassified;* to 11,114, *alcoholic hallucinosis;* to 12,196, *dementia precox.*
11,753	M.	40	*Alcoholic hallucinosis;* to 11,882, *delirium tremens.*
11,603	M.	60	*Alcoholic hallucinosis;* to 11,170, either *unclassified* or *cerebral arteriosclerotic insanity* or *alcoholic hallucinosis.*
1,188	M.	35	*Alcoholic hallucinosis;* to 1,703, *unclassified.*

1,736 M. 43 *Unclassified;* to 1,831, *alcoholic delusional insanity.*

Recoveries in persons in the third decade of life (21 to 30 years) were as follows:

11,007, 11,114, 12,196 F. 29 (1) *unclassified*, 32 days: (2) *alcoholic hallucinosis*, 61 days; (3) *dementia precox*, 25 days.
11,316 M. 21 *Manic-depressive insanity*, 79 days.
11,069 F. 29 *Alcoholic hallucinosis*, 8 days.
11,056 M. 29 *Alcoholic hallucinosis*, 36 days.
11,255 F. 28 *Alcoholic hallucinosis*, 49 days.
10,991 F. 21 *Dementia precox, catatonic*, 91 days.
11,119, 11,207 F. 21 (1) *manic depressive insanity*, 30 days; (2) *manic-depressive (manic phase)* 20 days.
911 F. 29 *Alcoholic hallucinosis*, 36 days.
946 M. 26 *Alcoholic psychosis*, 11 days.
958 M. 25 *Alcoholic psychosis*, 6 days.
969 F. 28 *Alcoholic hallucinosis*, 13 days.
1,022 M. 23 *Delirium tremens*, 16 days.
1,147 F. 24 *Alcoholic hallucinosis*, 30 days.
1,159 M. 27 *Alcoholic hallucinosis*, 27 days.
1,378 F. 24 *Alcoholic hallucinosis* (syphilis), 23 days.
1,425 1,633 M. 26 (1) *dementia precox, catatonic episode*, 16 days; (2) *dementia precox* (discharged unrecovered), 4 days.
1,774 M. 28 *Delirium tremens*, 19 days.
11,940 M. 28 *Delirium tremens*, 7 days.

Recoveries in persons in the fourth decade of life (30 to 40 years) were as follows:

11,073 M. 33 *Alcoholic hallucinosis*, 23 days.
11,124 M. 40 *Alcoholic psychosis*, 15 days.
11,371 M. 39 *Alcoholic hallucinosis*, 18 days.
11,413 M. 40 *Delirium tremens*, 14 days.
11,233 F. 35 *Alcoholic hallucinosis*, 26 days.
11,753, 11,882 M. 40 (1) *alcoholic hallucinosis*, 14 days; (2) *delirium tremens*, 11 days.
10,920 F. 35 *Exhaustion psychosis*, 24 days.
11,667 M. 40 *Delirium tremens*, 9 days.
12,266 M. 38 *Alcoholic hallucinosis*, 4 days.

870	M.	40	*Delirium tremens*, 11 days.
878	F.	34	*Alcoholic hallucinosis*, 10 days.
900	M.	40	*Alcoholic hallucinosis*, 1 month.
916	M.	37	*Alcoholic hallucinosis*, 18 days.
907	F.	35	*Alcoholic hallucinosis*, 1 month, 5 days.
938	F.	36	*Alcoholic hallucinosis*, 21 days.
967	F.	35	*Delirium tremens*, 16 days.
1,008	M.	36	*Delirium tremens*, 11 days.
1,014	M.	30	*Delirium tremens*, 7 days.
1,068	F.	38	*Alcoholic hallucinosis*, 12 days.
1,060	M.	33	*Alcoholic hallucinosis*, 10 days.
1,009	M.	35	*Delirium tremens* (*epilepsy*), 1 mon., 9 days.
1,188, 1,703	M.	35	*Alcoholic hallucinosis* or *classified*, (1) 18 days, (2) 4 days.
1,291	M.	31	*Delirium tremens*, 24 days.
1,278	M.	36	*Delirium tremens* (*epilepsy*), 8 days.
1,391	F.	38	*Alcoholic hallucinosis*, 22 days.
1,483	M.	40	*Delirium tremens*, 7 days.
1,532	M.	35	*Delirium tremens*, 11 days.
1,546	M.	37	*Alcoholic hallucinosis*, 5 days.
1,657	M.	40	*Alcoholic hallucinosis*, 10 days.
1,668	M.	40	*Delirium tremens*, 10 days.
1,739	M.	39	*Alcoholic hallucinosis*, 11 days.
11,116 11,926	M.	39	*Alcoholic hallucinosis*, (1) 17 days, (2) 13 days.
11,891	M.	36	*Delirium tremens*, 2 months, 13 days.
11,989	F.	40	*Alcoholic psychosis*, 20 days.

Recoveries in persons in the fifth decade of life (41 to 50 years) were as follows:—

10,845	M.	42	*Alcoholic hallucinosis*, 4 months, 3 days.
11,215	M.	47	*Alcoholic hallucinosis*, altered to *delirium tremens*, 12 days.
10,758	M.	42	*Alcoholic hallucinosis*, 1 month, 15 days.
11,592	M.	47	*Unclassified* (*alcoholic hallucinosis?*), 7 days.
11,434	M.	47	*Delirium tremens*, 2 months, 9 days.
11,557	F.	43	*Unclassified* (*depression*), 18 days.
11,858	M.	43	*Delirium tremens*, 12 days.
11,597	F.	42	*Alcoholic hallucinosis*, 1 month, 23 days.
888	M.	47	*Intoxication*, 1 day.

970 F. 41 *Delirium tremens*, 10 days.
985 M. 44 *Alcoholic hallucinosis*, 13 days.
1,021 F. 41 *Alcoholic hallucinosis*, 23 days.
1,047 M. 44 *Alcoholic hallucinosis*, 7 days.
1,062 M. 48 *Delirium tremens*, 21 days.
1,075 M. 45 *Alcoholic hallucinosis*, 1 month, 7 days.
1,230 F. 44 *Alcoholic hallucinosis*, 16 days.
1,454 F. 43 *Delirium tremens*, 6 days.
1,510 M. 41 *Alcoholic hallucinosis*, 1 month, 14 days.
1,686 M. 45 *Delirium tremens*, 16 days.
1,719 M. 44 *Alcoholic hallucinosis*, 12 days.
1,736, 1831 M. 43 *Alcoholic delusional insanity*, (1) 10 days, (2) 7 days.
1,841 M. 43 *Delirium tremens*, 29 days.
11,804 M. 50 *Delirium tremens*, 1 month, 16 days.
11,998 M. 41 *Alcoholic hallucinosis*, 9 days.
12,026 M. 45 *Alcoholic hallucinosis*, 10 days.

All of these fifth decade cases were alcoholic.

Recoveries in persons over fifty years of age were as follows:—

10,857 F. 71 *Delirium*, 4 days.
11,611 M. 58 *Delirium tremens*, altered to *alcoholic hallucinosis*, 20 days.
11,176 M. 58 *Alcoholic hallucinosis*, 1 month, 23 days.
11,203 M. 52 *Delirium tremens* (*pneumonia*), 15 days.
11,049 M. 54 *Manic-depressive insanity*, 28 days.
11,603, 11,770 M. 60 *Alcoholic hallucinosis*, 26 days; 11,770, either *unclassified* or *cerebral arteriosclerotic insanity* or *alcoholic hallucinosis*, 2 months, 2 days.
11,608 F. 63 *Delirium tremens*, 3 days.
12,274 M. 52 *Alcoholic hallucinosis*, 1 month.
915 M. 60 *Alcoholic psychosis*, 11 days.
956 M. 51 *Delirium tremens*, 29 days.
1,108 M. 52 *Delirium tremens*, 11 days.
1,190 M. 70 *Delirium tremens*, 16 days.
1,218 F. 52 *Alcoholic hallucinosis*, 6 days.
1,292 M. 51 *Delirium tremens*, 16 days.
1,191 M. 54 *Delirium tremens*, 1 month, 2 days.
1,555 M. 58 *Alcoholic hallucinosis*, 10 days.

12,010 M. 51 *Delirium tremens (general paresis?)*, 3 days.
12,004 M. 51 *Delirium tremens*, 13 days.

Thus, of these 18 recoveries in persons over fifty years of age, there were

Delirium tremens	9
Alcoholic hallucinosis, etc.	7
Delirium	1
Manic-depressive insanity	1

Recoveries outside the alcoholic group are as follows:

11,007, 11,114, 12,196, F. 28 possibly belongs in the alcoholic group, but was placed in her third attack in the dementia precox group.

10,857 F. 71 *Delirium*, 4 days.
11,316 M. 21 *Manic-depressive insanity*, 2 months, 19 days.
10,991 F. 21 *Dementia precox, catatonic*, 3 months, 1 day.
11,557 F. 43 *Unclassified (depressive)*, 18 days.
10,825 F. *Unclassified*, 2 months, 15 days.
10,920 F. 35 *Exhaustion-psychosis*, 24 days.
10,884 F. 25 *Morphine hallucinosis*, 28 days.
11,207, 11,119 F. 21 *Manic-depressive insanity*, (1) 1 month, (2) 20 days.
11,049 M. 54 *Manic-depressive insanity*, 28 days.
1,425, 1,633 M. 26 *Dementia precox, catatonic*, 16 days; discharged in second attack unrecovered, 4 days.
1,448 F. *Unclassified*, 13 days.

Future analyses may be made more thorough, and the elements of recovery may stand out more clearly in detail. Suffice it to say that a superficial analysis like the present amply proves several points.

First, the component of nursing cannot be omitted from these recoveries, brief as was the time of the hospital stay of the majority of cases. This is proved by the incidence of dis-

order of heat-regulation (fever, hypothermia) in at least 37% of the cases.

Secondly, the special value of nursing, and particularly of hydrotherapy, stands out from the results of the treatment of alcoholic psychoses, which though they form only about one-ninth of our problem of first care, represent almost nine-tenths of our early therapeutic results.

Thirdly, the recoveries in the so-called "recoverable" forms of insanity take too long to be represented in any numbers in this first hundred of recoveries, and it may be suspected that the average hospital stay of three to four weeks is not sufficient for recoveries in groups like manic-depressive insanity.

Fourthly, the effect of psychotherapy as applied in the Psychopathic Hospital, is not a rapid effect.

Fifthly, the percentage of syphilis in the recovered cases is exactly that of the total intake of the hospital, so that this factor cannot be said to influence treatment unfavorably (two questionable syphilitic cases are more fully discussed and reference made to Kraepelin's analysis of allied conditions).

Sixthly, some index of the activities of our after-care service is afforded by the fact that nearly half of the patients either resorted voluntarily or (in some cases) were brought to the out-patient department at one or more periods subsequent to the discharge.

Seventhly, the need is apparent of nurses who shall build their psychopathic training on a sound basis of general hospital work (letter quoted, to the committee of superintendents dealing with the general aspects of the nursing problem for the insane).

XXXIX

REPORT OF AN EPIDEMIC OF PARATYPHOID FEVER AT THE BOSTON STATE HOSPITAL, MASSACHUSETTS, 1910.

I. Epidemiological Features of an Outbreak of Paratyphoid Fever (Bacillus Paratyphosus Alpha).* By Myrtelle M. Canavan, M.D., Assistant Pathologist, State Board of Insanity, formerly Pathologist at Boston State Hospital.

II. Clinical Features of an Outbreak of Paratyphoid Fever. By Mary E. Gill-Noble, M.D., Assistant Physician, Boston State Hospital.

III. Notes on the Blood Cell Picture in Paratyphoid Fever and after Vaccination with Bacillus Typhosus. By Myrtelle M. Canavan, M.D.

IV. Note on the Relation of Paratyphoid Fever to Antityphoid Vaccination. Myrtelle M. Canavan, M.D.

V. Conclusions from Work on the Paratyphoid Epidemic at the Boston State Hospital, 1910. By E. E. Southard, M.D., Pathologist to the State Board of Insanity; Director, Psychopathic Department, Boston State Hospital; Bullard Professor of Neuropathology, Harvard Medical School.

I.

Epidemiological Features of an Outbreak of Paratyphoid Fever (Bacillus Paratyphosus Alpha).*

By Myrtelle M. Canavan, M.D., Boston,

INTRODUCTION.

This paper, together with four articles to follow, constitutes a report of work done with an epidemic of about 30 cases of a mild form of paratyphoid fever which occurred at the Boston State Hospital, between Oct. 19 and Dec. 1, 1910. There were no deaths. The epidemic was confined largely to employees, although a few patients were also affected.

The present report discusses certain general features of the epidemic, the possible foci of origin and distribution of the disease (with remarks on plumbing and food-storage), methods of prophylaxis and treatment, laboratory methods employed for differentiation of the organisms and some facts of clinical pathology.

The outbreak of paratyphoid fever, ushered in by 12 cases of illness (fever, aches, malaise), in October, 1910, was not recognized as such at once, or even as belonging to the typhoidal group of diseases. There had not been within

* This and the four following papers represent Contributions of the State Board of Insanity, Numbers 26-30 (1914.6-1914.10). The papers were presented in substance at the meeting of the Boston Society of Medical Sciences held at the Psychopathic Hospital, April 28, 1914. A preliminary report had been made at the April, 1911, meeting of the Assistant Physicians' Association of the State hospitals of Massachusetts, at the Boston State Hospital, (*Bibliographical Note.*—The previous contribution was by E. E. Southard, entitled "On the Topographical Distribution of Cortex Lesions and Anomalies in Dementia Precox, with Some Account of Their Functional Significance," No. 1914.5 [to be published in the *American Journal of Insanity,* October, 1914].)

ten years any similar outbreak. The disease was mild. Many cases gave such mild symptoms that they had to be excluded from the category of true disease of any sort. Sixteen further cases occurred in November and two more in December.

Before presenting charts illustrating certain features of the epidemic, a brief description of the hospital and of certain epidemiological factors will be presented.

DESCRIPTION OF HOSPITAL.

The Boston State Hospital, Dorchester Centre, Massachusetts, lies upon a triangular plot of land containing 232 acres, unevenly divided by Morton Street, east of which lies the older women's department, with seven buildings closely grouped, with a population of approximately the following in 1910: 475 patients, 80 nurses, 34 employees, 5 medical officers, which, with 24 non-resident employees, make up a total of 618 persons. The officers are lodged in various parts of the buildings. The resident employees have a separate eating place.

It is necessary to speak of the men's department, because, although the epidemic was largely in the women's department, there is a warrantable suspicion that the source of the epidemic may have been in the men's department. In point of fact, between 30 and 40 male patients go during the day to the women's department to work in various capacities, and they have their midday meal in the chapel dining-room of the women's department, returning at night to their respective wards in the male department.

The following table shows how far the epidemic was confined to the women's department:

Women's department, men ill 5
Women ill 23
Men's department, men ill 2
Women ill 0

With two exceptions, then, the epidemic was confined to the women's department.

The first indication of a disease of a typhoidal nature was obtained by the discovery of a positive Widal reaction in a female patient, No. 8276, assisting in the serving-room of the nurses' quarters. From the blood of this patient actively motile organisms were recovered. One of the male patients, No. 6227, who was an assistant in the preparation of food and had for one of his duties the turning of the meat machine, was found to have fever (101°); from this patient a Bacillus paratyphosus alpha was cultivated from the blood. Upon the discovery of these facts concerning the two patients just mentioned, the patients were removed from the kitchen and dining room and a variety of precautions were observed (Pasteurization of milk, washing of dishes in formaldehyde solution, disinfection of toilet rooms and floors, and systematic observation of temperature in patients and employees. The chef (Case L) later fell sick.

Six months after the epidemic, organisms of the Colon group and the Paratyphoid group were isolated from chopped meat which had been sent to the pathological laboratory for the purpose of making culture media. Thus stated, without cultural facts, it may be thought that the epidemic can be ascribed to infection of meat. The distribution of the bacilli in meat or other food in the women's department may well have been effected by the male patient, No. 6227. It is possible also, of course, that the patient

himself obtained the disease from previously infected meat.

Although, as just stated, it is probable that the source of the epidemic was infected meat, it was necessary in working up the data of the epidemic to consider various other sources of infection and methods of spread. The first in order is perhaps the plumbing. The water supply is from the Metropolitan system mains, and connection with the city sewage is maintained. There had been overflowing of toilets in certain basements and near the kitchen at times. No definite source of infection could be attributed to these toilets. They were immediately overhauled and set in good order.

In the past, numerous epidemics of various descriptions in various parts of the world have been attributed to conditions in the ground water. There had been a general overturning of earth at the hospital for the erection of a new laundry building and for the construction of a large ditch. Investigation could not reveal any connection between these operations and the epidemic.

The ice pond required consideration. This pond is between one-half and one acre in extent somewhat below the level of Harvard and Austin streets. Manure has an opportunity to infect the pond, and ice is regularly cut from the pond to store in the ice-house and in a similar structure near the kitchen at the main department, from which supplies are daily given out. The use of this ice was restricted immediately after the first cases appeared, and bacteriological examination was made of random samples by Dr. B. L. Arms and Miss E. Marion Wade of the City Board of Health. They reported these samples to be negative for the typhoid

group. It is less probable that the ice from the ice pond was the start of this epidemic than that the maintenance of infection of meat in the refrigerator, infected otherwise, should be responsible.

As for garbage and rubbish, they are handled under proper conditions by feeding to animals and by burning of dry materials.

Climatic conditions had to be considered. Dust from dumping ground for the town of Dorchester, adjacent to the hospital, as well as from ditching operations and laundry building excavation, laying open the earth about a stagnant pond containing an overflow of laundry water, was in evidence, and the wind during October blew in the proper direction to secure the blowing of this dust to the kitchen from the different sources.

It was also noted on all sides that flies lasted longer in the autumn of 1910 than usual; screening is for the most part adequate except for the main kitchen. There is no direct evidence connecting the flies with the source or spread of the epidemic, however.

With respect to extra-mural infection, it must be remembered that at this time practically every nurse and employee had returned from a two-weeks' vacation and that the mobility of nursing and employees' population is habitually extreme.

As for carriers, the fact that the chief engineer had been a victim of so-called typhoid fever in 1871-72 and still had certain symptoms of an abdominal origin had to be considered. This patient, however, was a victim of paratyphoid fever in this epidemic. The Widal reaction for bacillus typhosus in this patient was negative,

nor were organisms of the colon typhoid group grown from urine, feces or blood.

These investigations seem to leave as the best hypotheses for the origin and spread of this epidemic the statement made above concerning the infection of female patient 8276 followed by the infection of a male patient (6027) who had to handle the meat. The chef and the baker later fell sick, whereupon the epidemic was established.

The subsequent bacteriological examination of meat from the refrigerators seems to point to the possibility of persistent infection of this meat, although of course it is possible that more recently purchased meat might have been infected. It seems that meat is not, as a rule, preserved in the refrigerators for a period as long as six months. It is, therefore, probable that infection in the refrigerators, if it persisted, was local.

Below is given the autopsy protocol of Case 9586, which died some years after the epidemic, on the 26th of April, 1913. The protocol is presented because of the rarity of autopsy protocols in paratyphoid fever. The only indication of lesion in the body which can be safely or with probability attributed to the paratyphoid fever is a thickening of the ileum, but neither the jejunum nor the colon showed any gross signs. The gall-bladder failed to yield paratyphoid bacilli, as did also various other loci tested.

The organisms found were: heart's blood, micrococcus jongii; gall-bladder, streptococcus septicus; lumbar lymph nodes, bacillus coli communis.

Case No. 9586. Autopsy No. 1913-26. April 26. 1913. Head, trunk and cord: M. M. Canavan. H.P.M. 6. Age, 38 years.

Body of an obese white woman 138 c.m. in length.

Skin over trunk waxen white in color, over face and neck deeply blue red, which turns white on pressure to quickly return to blue on release of it; over the forearm occur small petechial red spots, and over the entire right leg and foot blue-red epidermis is seen, which separates from the corium, revealing a somewhat mottled subcutaneous shadow.

Both legs and abdomen and back muscles show marked pitting on pressure. There is a small decubitus over sacrum. No palpable lymph nodes. The hands are disproportionately small. Pupils, 0.6 c.m. in diameter and central and equal. The ophthalmoscopic examination at this time shows both nerve heads of dim outline; the vessels emerging from the right fundus are irregular in appearance and only here and there are visible; the veins from the left fundus are brilliant and wider than their fellows or the normal. Abdomen distended.

Ventral Section. Panniculus edematous and pale yellow in color. Measures 0.6 c.m. Muscles pale red in color. Peritoneal cavity contains much fluid, pale yellow. The intestines float in a great collection of this fluid. Spleen adherent to diaphragm. Appendix bound down, 6 c.m. in length. Ovaries and tubes adherent to the rectum and each other. No mesenteric lymph nodes palpable. Liver 8 c.m., stomach 18 c.m. below ensiform. Diaphragm arches to 4th rib on the right, 5th rib on left.

Thorax. Mammary vessels sclerosed. Pleuric cavities filled with fluid. Pericardial sac tense from great excess of contained fluid.

Heart. Distended. Weight, 415 grams. Epicardium scant of fat. Coronaries not notable. A patch of fibrin adheres to the apex of the heart. The heart muscle is red with grey streaks fairly firm. At the apex the wall is very thin. The endocardium is grey but no vegetations or thickenings occur; the free edges of the mitral slightly curled under, and the uncut valve admits four fingers. Measurements: T.V., 10.0 c.m. M.V., 12.0 c.m. P.V., 7.0 c.m. A.V., 7.0 c.m. L.V., 0.4-1-3 c.m. R.V., 0.3-

1.0 c.m. All four chambers contain large quantities of cruor clot. Arch of aorta free from sclerosis.

Lungs. Combined weight, 631 grams. Nothing of note except two small infarcts measuring 2 c.m. each in the base of each lung. Peribronchial lymph nodes not remarkable.

Organs of the Neck. Thyroid congested.

Aorta. Thin and elastic throughout. The right lumbar lymph nodes (draining right leg area) are edematous and large. Sacral lymph-nodes (under bifurcation aorta) not notable.

Abdomen. Spleen,—weight (with attached piece of diaphragm), 280 grams. 12 x 8 x 4 c.m. in measurement. Capsule thickened over superior surface and the hilus is adherent to stomach wall. Section shows much congestion; pulp firm, trabeculae not notable, malpighian bodies not numerous.

Adrenals. Very firm; otherwise not notable.

Kidneys. Weight, 340 grams. Perirenal fat edematous. Capsule of left not thickened nor adherent, right kidney capsule is adherent and thick. Cortex of left measures 0.8 c.m., of right, 0.4 c.m.-0.6 c.m. Left kidney drips blood on section, no cysts; the right contains less blood, cysts occur. Pelves negative.

Liver. Weight, 1530 grams. Edges blunt. Color deep purple blue. Section shows marked mottling from venous engorgement. Gall bladder distended. Contains no stones but is filled with grumous black fluid. The wall of the gall-bladder is thick (resisted knife); no puriform content.

Pancreas. Pink; otherwise nothing of note.

Gastro-Intestinal Tract. Stomach dilated, no rugae. The gastric mucosa everywhere seat of petechial hemorrhages and free hemorrhage is shown in mucus. Pylorus small. The duodenum and jejunum show congestion. The ileum is fairly free from congestion, but shows a slight thickening. No indication of ulceration, old or new. Colon free except for injection. (Paratyphoid in November, 1910).

Genito-Urinary Tract. Bladder injected. Vagina, short and grey; cervix blue, os patent with clear tenacious fluid exuding from it. Uterine body firm; contains two white fibromyomata, encroaching on the cavity of the uterus. Fallopian tubes tortuous and distended, ending about ovaries, to which and the rectum they are attached. Section of the right ovary shows fibrous change; of the left, many corpora lutea, no corpus hemorrhagicum.

Head. Hair fine, brown and fairly abundant. Scalp firm, considerable blood escapes on cutting it. Periosteum not remarkable. Skull thick, though plentifully supplied with diploe. Frontal, 0.4 c.m.-1.3 c.m.; temporal, 0.4 c.m.-0.8 c.m.; occipital, 0.7 c.m. An irregular endostosis of inner table of frontal bone has locally absorbed the dura mater. Pacchionian granulations in eight areas pierce the inner table of the skull, occasionally almost perforating the outer table. The dura is otherwise slightly thickened and the pia slightly over the vertex. There is a subpial hemorrhage over the right prefrontal pole and the depression of the pituitary body contains blood. The consistence of the cerebrum is unequal, the right parietal slightly softened. Considerable hemorrhage free at the base of brain. Vessels not sclerosed. The pons is minutely honeycombed with vacuoles (arteriosclerotic?). Olivary bodies firm. Convolutions show nothing of note. Optic nerve sheaths show considerable swelling near bulb, otherwise nothing of note. Ears free. The Gasserian ganglions and pituitary negative. Cord slightly soft and shows slight subdural hemorrhage over left lower cervical region. Brain weight, 1120 grams.

Anatomical Diagnoses. Cause of death and acute lesions: Cardiac; pulmonary infarctions; subpial and subcutaneous hemorrhages, acute fibrinous pericarditis; hemorrhagic gastroenteritis; sacral decubitus; mitral insufficiency; dilatation and hypertrophy of heart; chronic interstitial myocarditis; chronic passive congestion of liver, stomach, and intestines; general anasarca; interstitial nephritis;

internal mammary arteriosclerosis; chronic perisplenitis, chronic pericholecystitis; chronic ileitis (slight thickening); gastroptosis; lumbar lymphnoditis; chronic fibrous oöphoritis and salpingitis; fibromyomata of uterus; calvarium thick, frontal endostosis; excessive arachnoidal villi; adherent dura; chronic fibrous leptomeningitis (vertex, slight); right parietal region of brain soft; spinal cord slightly soft (6 hours post-mortem); hydrops nervi optici.

Cultures from gall-bladder, retroperitoneal lymph nodes, heart's blood, negative for organisms of colon-typhoid group.

Microscopic examination of the ileum showed that the gross thickening was due largely, if not entirely, to edema of the mucosa. No convincing evidence of chronic changes was found (some hyperlymphocytosis?).

II.

Clinical Features of an Outbreak of Paratyphoid Fever.

By Mary E. Gill-Noble, M.D., Boston.

The clinical course of the disease in the series of cases here presented was so irregular, and the symptoms so varied in the different individuals, that we have thought best to append a table in which each case can be followed separately and the frequency of occurrence of certain symptoms seen at a glance.

The cases are listed in the order of their occurrence, the letters of the alphabet referring to nurses and other employees, while the patients are listed under their case record numbers.

No. 8276 is a female patient who was employed in the serving-room connected with the nurses dining-room and who was suspected of

being the original focus of the epidemic, although she had absolutely no clinical symptoms at any time.

No. 6227 was a male patient employed in preparing meat and other food in the main kitchen.

Cases U and Y had had one and three inoculations, respectively, of typhoid vaccine.

It will be seen by reference to the table that the classical symptoms of typhoid, with the exception of epistaxis, were conspicuously absent. Several of the cases were so atypical or presented such a negative picture that they would not have caused any suspicion of a typhoid-like disease if they had not occurred during an epidemic.

The second nurse to be taken ill, however, Case B., was the one who presented throughout the most striking resemblance to true typhoid. For this reason blood was immediately tested (City Board of Health) for Widal reactions. All cases were handled as possibly typhoid, with precautions as to isolation, disinfection, diet, and treatment.

For eleven days all the reports from the Widal reactions were negative, as were the blood cultures taken from the ear.

Case B developed such marked chills, with high fever and profuse sweating, occurring on the third, fifth, and ninth days of her illness, that repeated examinations were made for a malarial organism. Also during this time four of the cases had mild sore throat, and one a severe bronchitis, which in the absence of positive typhoid evidence suggested epidemic influenza.

It will be noted that the leucocyte counts, which in most cases were taken more than once, were not diagnostic, being too high for a leucopenia, except in Case B, which was 2400.

On Oct. 30, eleven days after the first case was taken ill, two positive Widals were returned from the Board of Health, and immediately six cases were sent to the Boston City Hospital, since our own accommodations for infectious cases were very limited. Four were sent later, making ten in all.

Seventeen cases altogether were taken care of in the Boston State Hospital. Two men employees who lived outside were cared for in their homes by outside physicians.

No organisms of the colon-typhoid group were cultivated from the blood from the ear in 25 cases, until Dec. 3, 1910, fifty-five days after the beginning of the epidemic, when a B. paratyphosus (alpha) was obtained from No. 6227 and an allied organism from No. 8276 and Case J.

Nov. 11, four blood cultures were taken from the arm of the cases M, S, U, and No. 9586, and all of these yielded the organism closely resembling B. paratyphosus (alpha).

GENERAL SYMPTOMS.

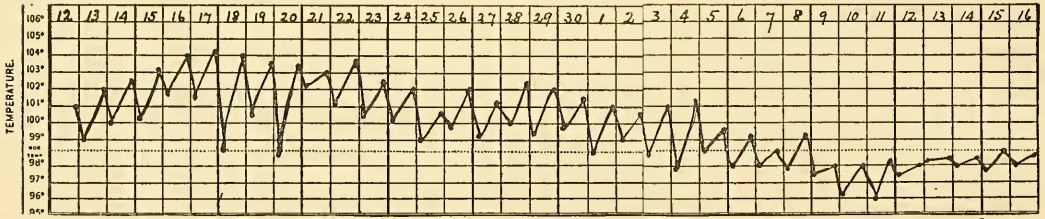
The onset in most cases was rather sudden, the prodromal symptoms seldom dating back more than a week from the day of discovery, and sometimes only a few days. Late in the epidemic, when it became a routine measure to take a daily evening temperature of all employees, several cases were discovered to have a slight rise to 99-100° without other symptoms. These were isolated and proved to be mild cases of the prevailing disease. A few of the severe cases had a more gradual onset and more marked symptoms. The duration varied from eleven days (Case P, in which fairly representative symptoms were present) to five weeks. Two had

a relapse and were in bed nine weeks, and another ran a continuous temperature for eight weeks and was in bed ten weeks with a long, slow convalescence following. This case recovered after a post-convalescent depression and returned to active service to die two years later of advanced tubercular involvement of the peritoneum.

The clinical charts followed no special law, there being almost as many types as there were cases. Only one showed the gradual steplike ascent peculiar to typhoid (Case V—chart reproduced). In several the evening temperature preserved the usual typhoid outline with deep remissions to or below normal either during the whole disease or throughout the first week. Others hovered around normal from the beginning, and two or three started in with the highest temperature the first two days, followed by a gradual and progressive descent to normal. The pulse was relatively slow, averaging from 70-108. The temperature and pulse given on the chart represents the highest point reached, and was usually observed only once or twice during the course of the disease.

The highest temperature of the twenty-four hours was usually registered between 10 p.m. and 2 a.m. The tendency in all cases was for the temperature to return to normal by lysis, but in several there was a sudden drop to or below normal, followed by an exacerbation to 101° or 102°, and then a more gradual and progressive descent. The condition of the patient at the time of this remission was suggestive of a crisis and a very noticeable improvement dated from that time.

Symptoms of the first week: Quite marked chills were noticed during the onset by many



CASE V. Temperature curve at the beginning suggestive of typhoid, but later dropping to normal, and then following a variable course.

patients, and several had repeated chills during the first week. Those of Case B, already referred to, were very severe and unusual. Sweating was also quite a common symptom, requiring for relief a sponge bath and change of bed clothes.

Headache was the most constant, and persisted in some cases more than a week, but in the majority disappeared the third or fourth, day, returning a few days longer with the evening fever.

Four had tenderness and rigidity of the posterior muscles of the neck. Cases B and M* complained bitterly of this, the latter obtaining no relief until the end of the second week.

There was very little insomnia. It was the rule for the patients to sleep well and to need very little attention at night.

Slight stupor was noticed in five cases, and mental dullness was present in the majority while the headache lasted; but even then they were remarkably bright and serene in the morning.

Two had slight difficulty in hearing for a day or two.

Twelve had markedly dilated pupils, sluggish in their reaction to light.

There was no delirium among those observed by us, with the exception of Case M, who wandered a little in her conversation one afternoon. Case B was somewhat delirious several days at the Boston City Hospital.

Pain in the back and limbs was general and was a striking feature, being more suggestive of the pain of influenza and tonsillitis than of typhoid.

Epistaxis occurred in nearly half the cases,

* For Case M, see Appendix.

but in some did not appear until the end of the week.

Sore throat existed in ten cases as a diffuse redness of the whole pharynx and fauces. Throat cultures were taken in all and were negative for the Klebs-Loeffler bacillus.

Bronchitis of a mild form occurred in six cases and was pronounced in one.

Rose spots were not found in any of the twenty-one cases we observed. Eruptions occurred in five cases, three being a diffuse papular eruption appearing in one crop and disappearing altogether in less than forty-eight hours, and the other two were an erythema of the face and neck, lasting about the same period.

The tongue as a rule showed white creamy coat, which persisted until the fever subsided and cleared gradually, beginning at the edges. Five had a brown furred tongue, but there were no fissures. There was very little tendency to the collection of sordes.

The spleen was enlarged to percussion in about one-third of the cases. It was palpable in only three observed by us.

Nausea and vomiting occurred in two cases during the first week and in one was persistent and troublesome for several days.

Diarrhea of the pea soup variety was observed for a day or two in four cases, and one patient had a slight attack several days before coming under observation.

Constipation prevailed and in two cases was so obstinate and troublesome as to cause some apprehension, repeated enemata and cathartics having very little effect.

The second week of the disease was an extremely comfortable period for the majority of these patients. Mentally they were noticeably

bright and cheerful; the constipation was under control; they were hungry and chafed at their restricted diet and enforced stay in bed. They felt so well that it was difficult to convince them of the wisdom of the recumbent position.

During this week five cases recovered, having been in bed altogether less than fourteen days, and of those who continued to run a temperature there was very little to note except an exhaustion out of all proportion to their symptoms and the severity of the fever.

A few had more severe symptoms.

Abdominal pain: It will be seen by the chart that only five cases had any abdominal pain whatever; one of these was very slight and another only complained for a few hours. Case M suffered extremely from the pain caused by enemata and cathartics, but was perfectly comfortable after her bowels had moved. She had stupes applied on one occasion.

Three had some flatulence which, however, was easily relieved.

Several others complained of tenderness of the abdomen on being moved or handled.

Five suffered from retention of urine on several occasions, which, however, yielded to hot applications.

Two had a sudden attack of cardiac pain with syncope in the second week. One of these was subject to neuralgic pain in this region. A third had pain and distress in the precordium throughout the first two weeks, but during a relapse of several weeks he was absolutely free from it.

During the third week only three cases had pronounced symptoms, and it was this group which appeared more like typhoid. The remainder continued to run a somewhat irregular

			Dur. of Prodrom.	Dur. of Illness.	Leucocyte Count.	Aggl. B. Typh.	Aggl. Para	Aggl. B. S. H.	Highest Temp.	Highest Pulse.	Epistaxis.	Sore Throat.	Bronchitis.
A.	Nurse	Oct. 19	1 wk.	9 wks.	7,200	+			103°	120	+	+	+
B.	Nurse	24	2 wks.	5 wks.	2,400	+			103	130	+	+	+
C.	Nurse	25	1 wk.	4 wks.	7,600				102	100	+		
D.	Nurse	26	2 wks.	4 wks.	8,000				104	128	+		
E.	Nurse	27	2 wks.	5 wks.	8,200				104	130			+
F.	Nurse	27	2 wks.	5 wks.	6,400				103.6				
G.	Nurse	27	1 wk.	4 wks.	4,800				103				
H.	Porter	29	1 wk.	8 wks.	6,700				104	120		+	
I.	Asst. Cook	31	1 wk.	3 wks.	4,200				104	120		+	
J.	Nurse	31	1 wk.	3 wks.	7,200			+	103		+	+	
K.	Porter	Nov. 1	3 d.		5,400				101	115		+	
L.	Cook	2	1 wk.	6 wks.	6,000				103.3		+		
No. 9586	Patient	2	1 wk.	5 wks.	7,000			+	104	138			
M.	Nurse	2	4 d.	7 wks.	6,000	+	+	+	104.4	120			
N.	Engineer	3	1 d.	6 wks.	4,200				104				
O.	Nurse	4	3 d.	12 d.	10,800								
					6,400				101	110		+	
P.	Nurse	5	5 d.	11 d.	5,000				101	104		+	
No. 6227	Patient	5	?	5 wks.	5,400		+				+		
Q.	Laundress	7	1 wk.	3 wks.	7,200				103.4	100	+	+	
R.	Nurse	8	1 wk.	9 d.	3,800				100.6	110			
S.	Nurse	8	2 wks.	6 wks.	7,600			+	103.8	118	+		
T.	Nurse	8	1 wk.	13 d.	6,800				103	118			
U.	Nurse	8	2 wks.	16 d.	5,800			+	103	120	+		
V.	Nurse	11	3 d.	4 wks.	5,200				104	130	+		
W.	Nurse	13	1 wk.	13 d.	3,200				102	108		+	
No. 8652	Patient	14	1 wk.	5 wks.	11,200								
					7,200	+	+		101.5	100	+		+
X.	Baker	17	1 wk.	4 wks.	4,800				102.8	115			
Y.	Nurse	30	2 wks.	10 wks.	5,200				101.8	108	+		+
No. 7779	Patient	Jan. 28	3 d.	4 wks.	10,200				102	108			
No. 8276	Patient							+	No clinical symptoms.				

	Chills.	Sweating.	Rash.	Coated Tongue.	Nausea and Vomiting.	Abd. Pain.	Abd. Tenderness.	Tympanites.	Diarrhea.	Constipation.	Enlarged Spleen.	Headache.	Stupor.	Delirium.	Dil. Pupils.	Deafness.	Stiff Neck.	Pain in Back.	Urine, Retention of	Praecord. Pain.	Stiff Joints.
A.	+	+	+	+	+	+		+		+	+	+	+	+	+	+	+	+			+
B.	+	+		+		+	+			+		+	+		+			+		+	
C.	+	+	+	+	+					+		+						+			+
D.	+	+		+			+		+	+		+						+			+
E.	+	+		+			+			+	+	+				+	+	+			+
F.	+	+		+			+			+		+						+			+
G.	+	+		+			+			+	+	+	+		+			+		+	+
H.	+	+		+						+		+							+		
I.	+	+	+	+						+		+					+	+			+
J.	+	+		+						+		+						+			+
K.	+	+	+	+				+		+		+						+			+
L.	+	+		+		+			+	+		+	+	+				+			+
No. 9586	+	+		+		+			+	+		+		+				+			+
M.	+	+		+						+		+						+			+
N.	+	+		+						+		+						+			+
O.	+		+	+						+		+						+			+
P.	+	+		+	+				+	+		+	+				+	+		+	+
No. 6227	+	+		+						+		+						+			+
Q.	+	+		+			+			+		+						+			+
R.	+			+						+		+						+			+
S.	+	+		+	+		+		+	+		+			+			+			+
T.	+	+		+			+			+		+						+			+
U.	+	+	+	+			+			+		+						+			+
V.	+	+		+			+			+		+						+	+		+
W.	+	+		+			+			+		+						+			+
No. 8652	+	+		+						+		+			+			+			+
X.	+	+	+	+		+				+		+			+			+			+
Y.	+	+		+			+			+		+						+			+
No. 7779	+	+		+						+		+						+			+
No. 8276	+	+		+						+		+						+			+

temperature with very little else of importance.

There were no instances of perforation or of hemorrhage. It is interesting to note that the menstrual function, so often disturbed in typhoid, was only suspended in one case, though rendered irregular in one or two more. Three cases following an attack of nausea and vomiting and some abdominal pain, had a relapse. In two of these the relapse was much more serious than the original attack. Case A was one of these, and up to this time, the twenty-ninth day, her only symptoms had been headache, slight constipation and moderate fever.

The last nurse to be taken ill ran a temperature for eight weeks, reaching 102° at night for six weeks of that time. The patient had no other symptoms except epistaxis, slight headache and constipation and was considered a very mild case, but on attempting to sit up she showed marked exhaustion, had several attacks of syncope, and was in a condition of mental depression for several weeks. She is completely recovered and on duty after a month's vacation.

COMPLICATIONS.

Were not severe. One patient, No. 9586, had lobar pneumonia; one, No. 6227, a very severe acute parenchymatous nephritis.

SEQUELAE.

With one or two exceptions all complained of stiffness of the knee and ankle joints after getting up, and one nurse after going home walked too far the first day and had an acute swelling of one ankle, which was painful and troublesome. Several of the nurses who were ill still complain at the date of writing of an unusual

tendency to fatigue, and shortness of breath on going up and down stairs.

TREATMENT.

Sponge baths were given once in three hours whenever the temperature rose above 102.5.° It was found very early that tepid baths were just as effectual in reducing the temperature as cold ones and were not dreaded by the patients.* Intestinal and urinary antiseptics were used to some extent. Enemata e. o. d. and sometimes cathartics for the relief of constipation. Very few hypnotics were needed. Aspirin was used somewhat for the headache. They were kept on a diet of liquids and milk, malted milk, beef tea, oyster and other broths, with occasional soft solids, such as cream toast, jellies, simple puddings, etc., and bread and potato with gravy for the milder cases.

The usual disinfection of bed clothing and excreta was practised rigidly from the start. Urine and feces were examined bacteriologically in many cases, with uniformly negative results.

APPENDIX.

Case M. Nurse, age 21. This was the most severe although not of the greatest duration of the cases remaining in the Boston State Hospital throughout her illness. She is reported in detail because it might have been more easily mistaken for true typhoid than any other under our immediate observation, and yet she was one of the two whose blood specimens gave a positive paratyphoid as well as typhoid reaction at the Board of Health Laboratory, and she was also one of the five to have blood cultures taken from the arm in which was

* Stimulants were employed in about half the cases, especially those who were ill longer than three weeks.

found the motile bacillus which Dr. Richards has described.

She was taken ill Nov. 2, 1910 about the middle of the epidemic, after only four days of feeling below par, and was on duty until ordered to bed because of high temperature. Her leucocyte count taken the second day of the disease, was 6,000. Her clinical chart is one of the four here reproduced.

During the first five days the prominent symptoms were chilliness, afternoon headache accompanying the rise of temperature, constipation, and pain and rigidity of the neck, chiefly of the left side. No epistaxis, no eruption. Her mental state was remarkably bright and clear. She slept six to eight hours a night and awoke so much refreshed in the morning that she would not believe that she had contracted the prevalent disease, and repeatedly asked if she could go on duty.

After the fifth day her temperature began to be higher in the morning, and from this time on she appeared very ill. She was troubled continually with the most obstinate constipation and suffered considerable discomfort from the necessary enemata and cathartics, but this always subsided when the bowels were relieved. On the eleventh day there was some distention and turpentine stupes were applied. This was the only time that any treatment for abdominal pain was necessary.

At the end of the second week she had lost much weight and showed marked exhaustion, appearing listless and depressed. She was still suffering from headache and the pain in her neck, of which she almost constantly complained. At this time she suffered on several occasions from urinary retention and obtained relief from hot applications.

On the fifteenth day she was delirious for a few hours. Her temperature was reaching 104° in the evening, she was having sponge baths every three hours, stimulants and salt solution by the Murphy drip and there was considerable anxiety concerning her condition. On the seventeenth day her temperature dropped suddenly to 96.2°, and although

it rose to 102.8° in the evening throughout the two weeks following, this drop marked a change for the better. The only event of importance during the fourth week was an attack of syncope following an attempt to sit up on the bed pan; she rallied quickly. On the third day of her normal evening temperature, the thirty-fourth of her illness, she sat up fifteen minutes. From this time on until her discharge from isolation two weeks later, she made a progressive and uneventful recovery. The only sequelae were a slight stiffness of the knees and ankle joints for a week and dyspnea on exertion.

Case 8652. This case is presented in contrast to Case M, as running a mild course, although showing a positive agglutination test for bacillus typhosus and atypical for bacillus paratyphosus alpha.

The patient was admitted for the third time Sept. 26, 1907, the mental diagnosis being dementia precox, paranoid form. Nothing in the early history of the case is of importance. During her illness, as

BLOOD-CELL PICTURE IN

Case.	Day.	Leucocytes.	Poly.		S. L.	
			No.	%	No.	%
Q.	1st	7200	800	69.68	242	21.20
P.	1st	5000	430	43.47	385	38.12
T.	2d	6400	500	49.65	315	31.34
*J.	2d	8000	700	63.56	253	22.96
*6227	2d	5400	318	57.49	145	26.11
*M.	2d	7000	800	66.21	285	23.72
R.	3d	3800	1000	76.27	208	15.77
O.	3d	6400	700	61.77	265	23.47
*S.	3d	7600	1000	65.2	325	21.2
V.	4th	5200	600	58.3	270	26.2
W.	4th	3200	600	47.2+	402	31.6
Y.	4th	5200	600	53.6+	315	28.1
†8652	4th	7200	3960	55.	1621	22.52
9	6th	6200	500	44.63	270	24.15
*6227	8th	....	793	69.99	179	14.09
X.	10th	4800	700	64.3	180	16.5
*U.	24th	5800	700	60.13	302	25.60
A.	29th	7200	453	54.38+	261	31.3+

* Motile bacillus recovered from the blood.
† 1709 cells counted.

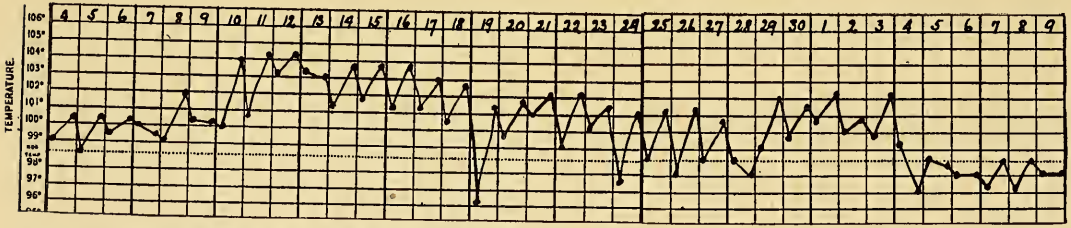
is frequently observed in similar mental states, she was quiet and coöperative, contrasting with active persecutory ideas to which she usually reacted.

The onset was at the end of the epidemic and after a week's irregular eating. There were no other prodromes, the first symptoms being nausea and vomiting in the morning, and slight nosebleed at night, with a temperature of 102.6°. For the first time, she complained of headache and general malaise. The following morning she had another slight nosebleed. On the third day of the disease, a diffuse papular eruption appeared over the abdomen, thorax, back, arms and thighs. There were about 125 in number,—diffuse red, fading the following day, and disappearing entirely at the end of three days, with no reappearance. The temperature during the entire course of weeks went to 100° or above only twice, ranging from 96.8° to 98.8°; the pulse-rate was correspondingly low, averaging 65. At no time was it dicrotic. Her respirations did not vary greatly, being about 20, and although

PARATYPHOID FEVER.

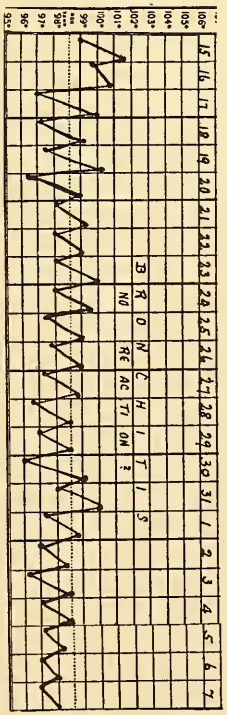
DIFFERENTIAL COUNT OF LEUCOCYTES.

L. L.		Trans.		Eosin.		Mast.	
No.	%	No.	%	No.	%	No.	%
58	5.05	44	3.70	2	0.10	2	0.10
109	11.02	57	5.7	6	0.6	2	0.2
60	5.97	75	7.46	55	5.4	..	...
80	7.2	48	4.3	2	0.18	..	...
50	9.0	41	7.4	..	...	..	...
85	7.05	30	2.4	..	...	5	0.4
64	4.87	32	2.5	..	...	2	0.1
89	7.84	70	6.17	8	0.70	1	0.09
138	9.0+	66	4.3+	3	.19+	3	0.19+
90	8.7	63	6.1	1	0.09	5	0.48
161	12.7	92	7.2	3	.2	13	1.0
100	8.9	85	7.6	11	0.9	7	0.6
960	13.34	577	7.1	7.2	0.1	36	0.5
150	13.41	191	16.10	7	0.60	6	0.50
57	5.02	98	8.6	6	0.5	..	...
95	08.7	108	09.9	..	...	4	0.30
92	7.90	54	4.60	14	1.20	2	0.10
82	9.80+	36	4.30+	..	...	1	0.10+



CASE M. Temperature curve in the most severe case paratyphoid fever. Note irregularity. Note characteristic drop to subnormal, 16th day (this drop occurs, as a rule, a little earlier, from 10th to 14th day). The pulse also drops at the same time. Subjective symptoms are most severe when the temperature and pulse drop. Case M also showed a species of recrudescence 10 days after the drop in temperature followed 6 days later by a second drop in temperature; but this second drop had no subsequent reaction by elevation of temperature.

during the second week she had a pronounced bronchitis, it did not change either the temperature, pulse or respirations. There were no abdominal symptoms, excepting constipation, which was easily controlled without producing diarrhea. The spleen was not enlarged. The most constant condition was white, coated tongue, which persisted well through convalescence. The blood showed leucocyte count of 11200 upon the second day, and at that time an agglutination test of a 1 to 50 dilution for bacillus-typhosus, and for bacillus paratyphosus alpha an atypical reaction. On the fourth, the leucocyte count was 7200 and the serum agglutinated bacillus typhosus was atypical for bacillus paratyphosus alpha. On the 25th day, the diet was gradually increased; she was up on the 35th day and placed on house diet.



Case 8652 (patient). Evening rise. Temperature over 100° but twice. Intercurrent (?) bronchitis failed to affect temperature.

III.

Notes on the Blood Cell Picture in Paratyphoid Fever and After Vaccination with Bacillus Typhosus.

By Myrtelle M. Canavan, M.D., Boston.

That there are specific infectious diseases which even at their height produce no hyperleucocytosis, differentiating them broadly from (*a*) certain other infections, and from (*b*) certain inflammations which demand surgical intervention, is accepted: Those which at their height do not send the leucocytes above their normal limits are: malaria, measles, typhoid fever, tuberculosis (uncomplicated), influenza, mumps, to which may be added bacillary dysentery[1] and paratyphoid fever.

If one could rule out in a given case malaria, tuberculosis, measles, influenza, mumps and bacillary dysentery, and were facing a differential diagnosis between typhoid and paratyphoid fever, the present work shows that the total count of leucocytes would be distinctly helpful. In typhoid fever a hypoleucocytosis (below 2000-4000 per cu. m.m.) is significant, combined with absence of eosinophiles. In this series of sixteen cases of paratyphoid fever the leucocytes were rarely below 4000 per cu. m.m. and the eosinophiles (found in differential counting of not less than 1000 cells) were present in a percentage of 0.31% (average of five cases in which the paratyphoid organism was recovered by blood cultures) and 0.8% in an average of eleven cases without culture (clinically certain epidemic cases).

[1] M. M. Canavan: Blood Cell Picture in Bacillary Dysentery, Boston Med. and Surg. Jour., Vol. clxi, No. 20, p. 62, Nov. 11, 1909.

AVERAGING THE CASES BY DAYS:

The first day showed...0.3 % eosinophiles (2 cases)
The second day showed.1.78% eosinophiles (4 cases)
The third day showed...0.29% eosinophiles (3 cases)
The fourth day showed.0.6 % eosinophiles (3 cases)
The six to tenth day
showed .....0.52% eosinophiles (3 cases)
The 20th to 30th day
showed .....0.6 % eosinophiles (2 cases)

It can be seen readily that the findings of the third day in this series most closely simulate typhoid hypo-eosinophilia. At the same time the leucocyte count rarely falls below 4000 (3800 in one instance on the third day, 3200 in one instance on the fourth day, these being in fact the only exceptions to the "over-4000" rule).

But, although paratyphoid fever does not show the characteristic typhoid hypoleucocytosis, paratyphoid fever cannot be said to produce hyperleucocytosis, since the counts never ranged over 8000. The leucocytes in paratyphoid fever in the present epidemic are consequently well within the normal range.

A chart shows the basis of these statements (see pages 24 and 25).

The chart shows the negative findings in other forms of cells. As a single procedure the leucocyte counting gives a suggestive lead in prognosis and diagnosis.

At the suggestion of Dr. Southard an attempt was made to count the leucocytes before and after antityphoid inoculations to see if an experimental cell picture resembling the changes in the disease of typhoid fever could be so produced. Too few cases were carried through enough counts to be conclusive, but there was an indication in one case (S. W. C.) in that the

leucocytes rose from mid-normal (6600) to higher normal (8000) in two hours after the first inoculation, and in fifteen hours to 10,000 with a drop in eosinophiles from 0.78% before inoculation to 0.72% two hours later, and 0.30% in fifteen,—imitating typhoid somewhat in the first days of the invasion (Table I).

In another case (S. E. V.) the leucocytes rose from 8400 per cu. c.m. to 9500 in two hours after the inoculation and were 8600 fourteen hours later; eosinophiles were also in evidence.

In another (M. E. G.) there was a rise after the first inoculation from 3600 to 4200 in twenty-four hours; eosinophiles also seen.

The fourth case (H. M. N.) presented a slight rise from 4400 per cu. m.m. to 6400 in four hours after inoculation, 3400 after eighteen hours, with an actual increase in eosinophiles from 2.4 to 3.9 (Table II).

The fifth case (M.C.) presented a slight rise in leucocytes from 5800 before inoculation to 7200 two hours after, with return to pre-inoculation number of 5800 per cu.m.m. in four hours, and an excursion of eosinophiles from 0.30-1.30-1.10% for the same number of counts (Table III).

All five cases presented, then, a slight oscillation upward in numbers of leucocytes after the first inoculation, but only one (S. W. C.) showed a tendency to disappearance of eosinophiles. The number of leucocytes differentially counted was high,—1000-5000 in each case.

TABLE I.—S. W. C.

Nov. 13, 1910, 7 P.M.
Before inoculation.
Leucocytes 6600
Polynuclears 62.80%

Small lymphocytes 22.05%
Large lymphocytes 4.40%
Transitionals 0.00%
Eosinophiles 0.78%

Inoculated with 0.12 cc. emulsion of typhoid bacilli killed at 53°, injected sub-cutaneously at insertion of left deltoid muscle.

Two hours later (after first inoculation).
Leucocytes 8000
Polynuclears 54.30%
Small lymphocytes 28.50%
Large lymphocytes 9.00%
Transitionals 7.20%
Eosinophiles 0.72%

Arm painful.

Nov. 14, 1910. 9.45 A.M.
Leucocytes 10,000
Polynuclears 81.00%
Small lymphocytes 7.50%
Large lymphocytes 5.40%
Transitionals 5.40%
Eosinophiles 0.30%

Dec. 4, 1910—Widal atypical.

TABLE II.—H. M. N.

Before inoculation:
Leucocytes 4400
Polynuclears 64.2%
Small lymphocytes 21.0%
Large lymphocytes 4.8%
Transitionals 6.7%
Eosinophiles 2.4%

Nov. 12, 1910, 5 P.M. 0.12 cc. emulsion of typhoid bacilli killed at 53°, injected sub-cutaneously at insertion of left deltoid muscle.

Four hours after inoculation:
Leucocytes 6400
Polynuclears 61.07%
Small lymphocytes 16.60%
Large lymphocytes 9.00%
Transitionals 7.60%
Eosinophiles 4.10%

11 A.M., Nov. 13, 1910. Arm painful.

Leucocytes	3400
Polynuclears	43.9 %
Small lymphocytes	37.8 %
Large lymphocytes	11.6 %
Transitionals	3.0 %
Eosinophiles	3.9 %

5 P.M. Nov. 17, 1910. Inoculated with 0.25 cc.
6 hours later, temperature 101°.
Headache lasting until 4 P.M. on the 18th.

Polynuclears	67.24%
Small lymphocytes	16.04%
Large lymphocytes	3.70%
Transitionals	10.30%
Eosinophiles	3.80%

Nov. 22, 1910.
Inoculated with 0.5 cc. emulsion.
(Widal atypical.)

Polynuclears	48.93%
Small lymphocytes	28.73%
Large lymphocytes	12.40%
Transitionals	10.30%
Eosinophiles	3.26%
Mast cells	0.50%

Nov. 27, 1910. 4.30 P.M.
Widal atypical.

Polynuclears	41.50%
Small lymphocytes	33.98%
Large lymphocytes	16.44%
Transitionals	4.29%
Eosinophiles	2.30%
Mast cells	1.80%

Inoculated with 0.75 cc. emulsion of typhoid bacilli.

Nov. 28, 1910.
Headache vertex and occiput.
Dec. 7, 1910. Widal positive (dilution 1-50).

TABLE III.—M. M. C.

Before inoculation—10 A.M., Nov. 7, 1910.

Leucocytes	5800
Polynuclears	51.68%
Small lymphocytes	37.12%

Large lymphocytes 6.29%
Transitionals 3.40%
Eosinophiles 1.30%
Mast cells 0.90%

99.89%

Inoculated 6 P.M. with 0.12 cc. emulsion of typhoid bacilli killed at 53°, injected sub-cutaneously at insertion of left deltoid muscle.

8 P.M.
Leucocytes 7200

2 A.M. Chill.
Nov. 8, 8 A.M.
Leucocytes 5800
Polynuclears 64.78%
Small lymphocytes 23.15%
Large lymphocytes 5.90%
Transitionals 3.24%
Eosinophiles 1.10%
Mast cells 1.00%

Marked malaise. 99.17%

Nov. 12, 1910. 8.30 A.M.
Leucocytes 4200
Polynuclears 41.34%
Small lymphocytes 43.38%
Large lymphocytes 7.63%
Transitionals 3.05%
Eosinophiles 2.50%
Mast cells 2.03%

99.48%

Inoculated with 0.25 cc. emulsion.
Chilly 8 P.M.

Nov. 12, 1910, 9.30 P.M.
Leucocytes 6200
Polynuclears 50.68%
Small lymphocytes 42.20%
Large lymphocytes 3.17%
Transitionals 1.90%
Eosinophiles 1.20%
Mast cells 0.80%

99.95%

Nov. 13, 1910. 12.30 A.M.—2. AM.
Chills.
11 A.M.

Polynuclears	62.78%
Small lymphocytes	26.90%
Large lymphocytes	2.06%
Transitionals	6.18%
Eosinophiles	1.61%
Mast cells	0.40%
	99.93%

Nov. 17, 1910. 11.30 A.M.

Leucocytes	6400
Polynuclears	42.67%
Small lymphocytes	45.26%
Large lymphocytes	4.80%
Transitionals	4.08%
Eosinophiles	2.04%
Mast cells	1.10%
	99.94%

Inoculated with 0.5 cc. emulsion.

Nov. 22, 1910.

Polynuclears	50.21%
Small lymphocytes	38.39%
Large lymphocytes	5.60%
Transitionals	4.40%
Eosinophiles	0.60%
Mast cells	0.70%
	99.90%

Widal atypical.
Dec. 7, Widal positive (dilution 1-50).

CONCLUSIONS.

1. Leucocyte counts may prove of some service in differentiating paratyphoid from typhoid fever, since counts show that the paratyphoid blood picture remains within the normal range (*a*) without tendency to hypoleucocytosis at any stage of the disease, and (*b*) without loss or drop in eosinophiles.

2. In interpreting this result it must be remembered that the epidemic from which these data were derived was one of mild paratyphoid fever (bacillus paratyphosus, alpha type).

3. The blood cell picture after antityphoid vaccination remains within the normal range, with tendency to slight initial rise in leucocytes (one instance only of a slight drop in eosinophiles).

IV.

Note on the Relation of Paratyphoid Fever to Antityphoid Vaccination.

By Myrtelle M. Canavan, M.D., Boston,

During the Boston State Hospital epidemic of paratyphoid fever (1910), the hospital administration instituted all precautions suggested by the Boston Board of Health, as well as various other precautions, to stop the spread of the disease. No attempt was made to inoculate patients, since but few had been attacked; but antityphoid vaccination was offered the 143 resident and non-resident physicians, nurses and employees, of whom 61 received one or more inoculations, and 50 received the prescribed four inoculations (5-day intervals,—25, 50, 75, 100 millions bacilli, prepared by Dr. Leslie H. Spooner of the Massachusetts Board of Health).

Of the 50 inoculated, no one contracted typhoid; and but one contracted paratyphoid fever. This one, a nurse (A. P.), fell ill six months after the epidemic was over, and from the blood Bacillus paratyphosus, alpha type, was recovered, the disease running a three weeks' course and terminating in complete re-

covery. In this case the antibodies produced by B. typhosus were obviously not effective against B. paratyphosus, alpha.

Of the eleven persons who received partial vaccination (from one to three inoculations), one was reported (three inoculations) to have had a typhoid-like disease (possibly paratyphoid; data lacking) three weeks after leaving the hospital; Case U (one inoculation) contracted paratyphoid (fever on evening of vaccination) and the organism (alpha type) was recovered from the blood; Case Y was ill ten weeks with a continuous fever, no culture taken. This patient died in July, 1912, of tuberculous peritonitis, and should probably be regarded as a case of unrecognized tuberculosis.

Of the 82 uninoculated physicians, nurses and employees, 12 were sick before the vaccination was started. Of the remaining 70 (unvaccinated), 18, or 26%, became sick (in the epidemic).

In this connection, I may refer to the work of Firth,[1] of the Royal Army Medical Corps in India. Firth found that the influence of antienteric inoculation against infection with paratyphoid fever was apparently nil. At any rate, of 104 cases of paratyphoid fever in 1911, it appears that 97 had been inoculated against typhoid fever and 7 not inoculated. They naturally suggest a bivalent emulsion.

1. Of 50 antityphoid vaccines exposed to paratyphoid fever in dining-rooms and otherwise, prior to and during an epidemic (Boston State Hospital, 1910), one case contracted paratyphoid fever six months after the epidemic (B. paratyphosus, alpha).

2. Of 11 antityphoid (incomplete) vaccines,

[1] Firth: Jour. Roy. Army Med. Corps, No. 19, p. 157, 1912.

one showed first symptoms of fever on evening of inoculation (disease already incubating; B. paratyphosus, alpha); one may have had paratyphoid fever (data lacking); one had symptoms, presumably tuberculous.

3. Of 70 persons not subjected to antityphoid vaccination, but equally (or rather less) exposed to paratyphoid fever with the 50 vaccines, 18 (or 26%) contracted paratyphoid fever (epidemic cases, four with positive bloods, the only cases in fact from whom blood cultures were taken among the 18).

4. Possibly these findings indicate a degree of "crossed" protection (typhoid vaccine against paratyphoid fever); but this is not absolute, and consequently paratyphoid vaccination is indicated in appropriate circumstances.

V.

Conclusions from Work on the Paratyphoid Epidemic at the Boston State Hospital, 1910.

By E. E. Southard, M.D., Boston,

As pathologist to the Danvers State Hospital, 1906-9, and later as pathologist to the State Board of Insanity, I have had repeated occasion to supervise or give advice in epidemics. The work in some of our Massachusetts state hospitals for the insane in connection with these epidemics has often threatened to put an end to all other forms of scientific laboratory, as well as clinical, activity. Yet I have always held that the scientific laboratories of the state hospitals should be built around what may be termed a hygienic nucleus, and that after all our first

duty as physicians in these hospitals is a duty to medicine at large and our second duty that to our specialty, mental disease, as such.

I may quote from my reports as pathologist to the State Board of Insanity of 1909 and 1910 in this connection as follows: —

"With respect to laboratories, the experience of our own as well as that of the general hospitals demonstrates that the most effective and lasting upgrowth of scientific spirit means building up the laboratory about a hygienic nucleus.

"In the first place, our patients require, for humanitarian and economic reasons, the best hygienic care,—care which nowadays indispensably calls for laboratory devices. By the routine performance of laboratory tests, which answer questions relating to the individual patient, and react on all the patients of an institution, the laboratory staff acquires the confidence of the ward staff, and the problems of each become the problems of the other. Thus, during the supervision of epidemiological work at Danvers Hospital, on bacillary dysentery, I noticed a progressive drawing-together of the laboratory and ward staffs. Besides the epidemiological results, which depend largely on recording what had hitherto remained unrecorded, the institutions actually obtained increased knowledge of their patients. During epidemic or endemic conditions, it was formerly the case that a tremendous amount of work with individual patients was done (though little recorded). and that psychiatric work was reduced to the barest necessities. Nowadays, with the foresight obtained from proper coördination, and the results of laboratory tests, even a diphtheria epidemic may not signify the stoppage of all efforts to classify patients accurately, and to accord to appropriate patients their individualized treatment. Moreover, the laboratory apparatus and recording facilities sufficient for hygienic work and the ordinary tests of general medicine make, with rela-

tively inexpensive additions, an effective laboratory for psychiatric work." (*Report of 1909.*)

"The hygiene of State hospitals for the insane bids fair to be a subject of the greatest importance in the progress of public health work in general. Though not obvious at first sight, it is true that no other conditions are so favorable for the exact study of epidemics in man as the conditions of (*a*) regimen, (*b*) surveillance, (*c*) variety of life, (*d*) most important of all, presence of laboratories and laboratory workers found in modern State hospitals. Elsewhere it has always been possible for hospitals to secure special government workers or some local enthusiasts to work with precision in these epidemics; but neither the origin nor the subsidence, and especially not the interepidemic conditions, have been studied so well as in Massachusetts, whose State hospital laboratories have been constructed about a hygienic nucleus. In the sense that the first apparatus installed has been apparatus of equal service in hygiene and in psychiatry, and the pathologists have been imbued with the salutary modern idea that no sharp lines separate psychiatry from the rest of medicine and hygiene. Therefore, although a certain amount of energy has been spent by our pathologists on hygienic problems, I have felt the energy well spent, and have encouraged the ideal of the hygienic utility and advantages of our hospitals." (*Report of 1910.*)

One advantage of writing out the conclusions of a piece of work in which one had so little immediate personal share is that one can duly praise the endeavors of the actual workers. Especially is the work of Dr. Mary Gill-Noble, then Dr. Gill, to be praised for its fidelity and insight, the latter rendered doubtless keener by her previous prolonged and confining work with a diphtheria epidemic in the same hospital. In the rush of clinical work, one so often hears the worker say, "Why, doctor, we are so busy

doing work that we have no time to record it," that the scientific attitude of actually recording clinical findings under epidemic conditions in hospitals chronically undermanned is deserving of great praise.

As for the laboratory side, the previous experience of Dr. E. T. F. Richards and Myrtelle M. Canavan in the Danvers dysentery epidemic stood them in good stead on the present occasion, and I need only point out, what is not always understood by those not directly in the work, how difficult it is to carry on special researches on top of regularly established routine. There were six medical workers in connection with the scientific side of the Danvers dysentery epidemic of 1908: here there were but three (or four, if the writer be counted), a quota far fewer than any hygienic commission would choose for such a purpose. At the risk of elaborating the obvious, I wish to point out that, in decades to come, our successors will wonder at our niggardliness in the comparatively inexpensive matter of research both in hygiene and in psychiatry. One reason for working overtime now is to lay a basis for adequate state support of these research matters in future. Yet I suppose there are many administrators of hospitals, even in the most highly civilized portions of our country, who would smile a superior smile at the idea of employing one or two additional skilled workers in epidemiology at the first appearance of an epidemic in a state hospital. "Why, doctor, what is our laboratory for, if not to meet and handle just such conditions as those of an epidemic? Why hire additional workers? Simply let the ordinary routine go for a time!"

I can the more safely indulge in these mild

platitudes because, after all, in the present epidemic perhaps not much was sacrificed to the Procrustean ideal of not raising the per capita cost. And the epidemic happened to be of no great proportions. In future, however, it seems to me that either the hospital itself or the state board of insanity should be empowered under epidemic conditions freely to call in additional skilled and technical aid, or, if this is not feasible, that reasonable expenses of such scientific epidemiology should not be frowned upon if they appear in the form of a deficit from the annual appropriation.

GENERAL CONCLUSIONS.

Etiology. The microörganism of the epidemic, according to Richards' findings, is undoubtedly Bacillus paratyphosus (agreement in general features with known bacilli; differentiation from Bacillus typhosus and Bacillus coli communis by growth on carbohydrate media).

The type is very probably alpha or acidumfaciens (behavior in litmus milk, agglutination reactions). This fact is of interest since it conforms to the general rule that the alpha type is more frequent in this country than the beta or alkalifaciens type. But it is curious that it is the beta type which is ordinarily supposed to be the "meat-poisoning" organism, whereas here we are apparently dealing with a "meat-poisoning" epidemic due to the alpha type.

Epidemiology. Circumstantial evidence seems to point to the immediate origin of the outbreak from a male patient who had been a kitchen-assistant occupied with preparing meat, developed fever, and yielded what was eventually de-

termined to be Bacillus paratyphosus in his blood.

At the same time a woman patient developed fever, a Widal reaction (typhosus), and actively motile organisms in her blood. This patient helped to serve food.

Appropriate sterilizing measures were carried out in the kitchen and serving-room, but cases continued to develop during the succeeding month.

A question which must remain unanswered is the method by which the first patient became infected. He had been slightly ill for several days before becoming febrile, but, being hypochondriacal, he secured little attention for his complaints. Whether he infected the meat (some of which six months later showed Bacillus typhosus) or was infected by it cannot be safely asserted. The absence of other similar cases before the epidemic might point to some extramural source (*e.g.* new attendant, or nurse returned from vacation, or infected meat); but, on the other hand, if the organism persisted in the ice-box for six months, there were no further cases of disease which could be assigned to the organism, except one nurse who had a moderately severe attack (B. paratyphosus alpha in blood) in June, 1910.

The season in which the outbreak developed is a little later than that in which the majority of typhoid fever patients are admitted to hospital, but the epidemic ceased with the advent of colder weather (December). The previous season had been hot and dry, and the groundwater was low—conditions thought to favor the development of typhoid fever.

In this small epidemic, 23 women and 7 men were affected, thus reversing the figures for the

development of typhoid fever, in which males far outnumber females.

The fact that patients were largely spared may perhaps be due to the greater age of patients, *i.e.* if conditions in paratyphoid fever resemble those in typhoid fever, in which there is a very decided drop in the incidence after forty years of age.

Dust, flies, ice (from local ice-pond), plumbing, refrigerators, were considered as factors in the outbreak. Beyond noting several features in this old plant which require alteration, the writers were not able to incriminate any of these conditions as the factor in the outbreak. Possibly infected meat was the source.

It appears most instructive to compare the findings of this epidemic of paratyphoid fever with those of typhoid fever. The results may be presented in parallel columns with some comments.

Typhoid Fever. (T. McCrae, 1913)	Paratyphoid Fever. (Data of this epidemic, Gill-Noble.)
Onset most frequently	Same.
1. with headache (1117 in 1500 cases)	
2. Anorexia (825)	Not marked, as a rule disappearing in second week.
3. Diarrhea (516)	4 cases in 30.
4. Abdominal pain (443)	5 (2 of these slight) in 30 cases.
5. General malaise (436)	Not severe.
6. Vomiting (404)	2 cases in first week.
7. Cough (393)	Mild bronchitis in 5 cases, (severe in 1)
8 and 9. Chilly sensations (392) and chills (334)	15 of 30 cases showed chills.
10. Epistaxis (323)	13 of 30 cases, but often not initial.

11. Fever (285)	Frequent, low.
12. Nausea (255)	2 cases (combined with vomiting).
13. Constipation (249)	Persistent in 2 cases.
14. Backache (215)	General (suggesting influenza or tonsillitis).
15. Sweating (207)	Common (20 cases in first week).

The onset of symptoms in this epidemic accordingly presented a few minor differences from that pictured by McCrae in the left hand column above. The onset symptoms, merging in some instances into symptoms of the first week, appear statistically in the order: headache (29), backache (27), sweating (20), chills or chilly sensations (15), epistaxis (13). As for initial fever, the later experience with routine temperature-taking in all "contacts" seems to show that initial fever was practically constant. Anorexia, diarrhea, abdominal pain, general malaise, vomiting, were not so frequent in this paratyphoid epidemic as in McCrae's combined Johns Hopkins figures for typhoid fever. The figures for the remainder of the fifteen major onset symptoms of typhoid fever listed above are too small for effective correlation.

McCrae mentions, for typhoid fever, six special modes of onset, of which the second (pulmonary) is perhaps the only one illustrated in the paratyphoid epidemic (bronchitis in five cases). One patient had lobar pneumonia.

Typhoid Fever. (T. McCrae.)	Paratyphoid Fever. (Data of this epidemic, Gill-Noble.)
Symptoms of second week more severe.	5 of 30 cases recovered completely in second week; in others only symptom, disproportionate exhaustion.

Early general appearance frequently characteristic.	Negative or not characteristic.
Fever constant, 1118 in 1500 reached 104°	Same.
	8 in 30 reached 104° (only one reached 104.4°).
Chills not infrequent.	Same.
Skin: rose-spots (80%)	No rose-spots in 21 cases specially examined, but eruptions in 5 cases (3 papular).
Sweating not common at onset and rare in course.	Sweating in two-thirds of all cases.
Diarrhea in course (17.4%)	Diarrhea (16.6%).
Constipation in course (51%)	Constipation (76.6%).
Abdominal pain (44%)	Abdominal pain (16.6%).
Hemorrhage (7%)	Hemorrhage (0).
Perforation (true % not yet established—2-3%)	Perforation (0).
Spleen felt (71.6%)	Spleen enlarged (33%) but palpated in 3 cases only.
Bronchitis "almost a specific symptom."	Bronchitis (16.6%).
Lobar pneumonia (1.5%)	Lobar pneumonia 1 case in 30.
Bronchopneumonia (1.2%) London fever hospitals.	Absent.
Pulse "less than would be expected from fever."	Pulse averaged 70-108.
Heart not characteristic.	Precordial pain, 3 cases (2 with syncope in second week).
"Nervous" symptoms of some sort almost universal.	Headache, 29 cases; stupor, 6 cases; delirium, 3 cases; dilated pupils, 12 cases.
Deafness without discoverable lesion comparatively frequent.	Deafness 2 cases.
Retention of urine common at onset.	Retention (16.6%).
Acute nephritis hardly 1% (Curschmann)	1 case (insane).

Menstruation as a rule ceases.	Suspended in 1 case.
Arthritis rare.	Stiff joints (19 cases in 30).
Duration 29.4 days of fever, 49 days from onset to discharge.	Duration 11 to 35 days.

The laboratory findings may be grouped as follows:—

Typhoid Fever. (T. McCrae, 1913.)	Paratyphoid Fever. (Richards and Canavan).
Bacilluria, probably 20-25%.	Not found in cases examined.
Comparatively few bacilli in stools.	No *paratyphosus* found.
Bacillemia 75%, (Coleman and Buxton).	Bacillemia, 100%, of cases examined (7), 2 as early as third day, 2 on ninth day.
Widal reaction almost always present at some stage, rarely before seventh or eighth day).	Widal reaction (*Bacillus typhosus*) positive, 4 cases in 30 (one of the 4 yielded *bacillus paratyphosus*), suggesting "group-reaction."
Leucopenia, to some extent progressive with the severity and duration of the disease.	Bacilli clumped by immune sera (*paratyphosus alpha;* less marked, *paratyphosus beta;* not *typhosus.*
In case there is suspicion of paratyphoid fever, blood cultures necessary or tests with patient's serum against his own bacilli.	Tendency to leucopenia, hardly of diagnostic value, except as excluding some forms of sepsis.

GENERAL CONCLUSIONS.

The epidemic of mild paratyphoid fever at Boston State Hospital in 1910 seems beyond

question due to Bacillus paratyphosus, alpha (findings of Richards)[1].

Apparently the source of the epidemic was infected meat or else a patient with paratyphoid infection may have spread the epidemic through meat. The clinical features of the epidemic of greatest interest are presented in the above parallel columns. Mention may be here made of the fact that initial fever was practically constant. Anorexia, diarrhea, abdominal pain, general malaise and vomiting were not so frequent in this paratyphoid epidemic as they usually are in typhoid fever. Of special modes of onset which might be confusing in diagnosis, bronchitis may be mentioned. In the course of the disease stiff joints occurred in 19 of 30 cases of paratyphoid fever, whereas in typhoid fever, arthritis is rare. Four of the patients suffered from muscle pains in the back of the neck. Malaria was suspected in one case from the nature of the acute symptoms.

A case is presented which died some years after the epidemic and showed thickening of the ileum, possibly representative of former intestinal disease, but whose cultures, including that of the gall bladder, failed to show bacillus paratyphosus.

The clinical picture shows points of interest in the temperature curves. A case will occasionally have a beginning temperature suggestive of typhoid fever, but this is not the rule. The intercurrent bronchitis fails to affect the temperature reaction, so that it may possibly be supposed that it is a portion of the disease rather than a truly intercurrent phenomenon. But it must be remembered that insane patients

[1] E. T. F. Richards: Bacteriology of Epidemic of Paratyphoid Fever, Journal Lancet, St. Paul, 1913.

show some differences in fever reactions to various infections from the reactions shown by sane persons.

Eight cases in 30 showed a peculiar drop of temperature to subnormal in the second or third week,—as a rule, between the tenth and fourteenth day. This temperature drop is accompanied by a drop in the pulse and by an access of the most severe subjective symptoms felt at any time in the disease.

The blood cell pictures show that there is no hypoleucocytosis or loss or drop in eosinophiles in paratyphoid fever (of the type here described), since the counts remain within the normal range; this point may be of some value in differentiation of paratyphoid fever from typhoid fever.

It was incidentally learned that the blood cell picture after antityphoid vaccination also failed to show hypoleucocytosis, but instead tends to show a slight initial rise in leucocytes.

The antityphoid fever vaccinations carried out early in the epidemic as a possible protective measure, gave the opportunity for a few observations on the relation of paratyphoid fever to antityphoid vaccinations, and it would appear that there may be a moderate degree of crossed protection (typhoid vaccine against paratyphoid fever) since non-vaccinated persons were far more subject to paratyphoid fever than were persons vaccinated against typhoid. The rule was, however, not absolute.

XL

ON THE NATURE AND IMPORTANCE OF KIDNEY LESIONS IN PSYCHOPATHIC SUBJECTS: A STUDY OF ONE HUNDRED CASES AUTOPSIED AT THE BOSTON STATE HOSPITAL.*

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AND

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Following is a study which grew out of a superficial inquiry into the topic of insane hospital dietaries, dealing more particularly with the question whether said dietaries can properly be arranged on the same principles as dietaries for a group of sane persons, such as soldiers, prisoners, and paupers. Our experience in the post-mortem rooms of hospitals for the insane has left us with the general impression that various organs having intimately to do with metabolism, and notably the kidneys, are found subject to disease in proportions so high that the scientific dietician must perforce take them into account. In the present study, however, we draw no dietetic conclusions from our kidney findings, and leave the topic with the word that the proportion of serious renal disease in the insane is too high to be safely neglected in the dietetics of insane hospitals, and this despite the possibility that age and other complicating factors may elevate our proportions of renal disease unduly. If it be urged, or even proved, that a similar age-group drawn from

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almshouse material would yield as high or almost as high proportions of renal disease, the dietetic conclusion would be all the more important. We hope that some day some one will study almshouse material with equal fidelity. This may happen when chronic disease becomes as serious an object of study as acute.

It is worth while to print here some remarks made at a recent semi-annual conference of the State Board of Insanity of Massachusetts with the trustees and superintendents of insane hospitals[1]:

"Statements have been made to the effect that diets for the insane probably differ in no important respect from diets for the sane. This, I think, is not entirely the case. In the first place, the majority of our patients are in advanced years and possibly subject to metabolic disorder either connected with the advance of age or with mental disease.

"I therefore wish to make a few points concerning the theoretical necessity of more work on special diets for the insane.

"First, the victims of mental disease are unusually prone to kidney disease. When the subject of diet in mental disease came up for this conference, it occurred to me to look up the figures for kidney disease, expecting to find a high percentage on account of the advanced age of many of our patients. I was hardly prepared to find that about seventy per cent of all autopsied cases showed chronic Bright's disease in some form.

"My figures are derived from random consecutive autopsies at the Danvers and Boston State Hospitals to the number of 1,030, examined from 1902 to the present time. Six hundred and ninety-three of these cases showed some form of chronic nephritis, and, if the list were swelled by inclusion of various rare forms of chronic kidney disease, it would not be hard to build up a percentage much higher than seventy.

"What is the significance of these figures? The majority of our patients come to autopsy in advanced years Some authorities say that every person over fifty years of age has some evidence of chronic renal disease. If so, a portion of our percentage is accounted for. Another possibility is that the renal disease is a terminal condition which does not run parallel with the mental disease and is either a late complication of the mental disease or is entirely independent of mental disease. The majority of those cases dying under forty and characterized by chronic Bright's disease are victims of general paresis; here it is difficult to avoid supposing that both the brain disease and the kidney disease have common causes.

"Whatever be the cause of the high proportion of renal disease in our patients it is clear that the problem is deserving of study and that an exact clinical study might yield dietetic conclusions of interest. Such

exact clinical study is well nigh impossible under the conditions of hospitals for the insane, since no one seems to know how to procure the twenty-four hours amount of urine which is a desideratum for all standardized work. At least, this appears impossible for more than the occasional case with the number of nurses and attendants available.

"Secondly, the same autopsy series mentioned above yielded 132 cases of acute Bright's disease, or about thirteen per cent. It is more within the bounds of possibility to make this diagnosis in life from small samples of urine and other signs. Still it is a question whether there has been adequate attention paid to the matter of special diets in this group of cases.

"Thirdly, Dr. Earl D. Bond, lately clinical director and pathologist at Danvers State Hospital, has called my attention to observations of his tending to show that glycosuria on admission to hospital is perhaps very often a matter of inanition.

"Fourthly, still another matter which suggests further study of the question of special diets is the frequency of gastro-intestinal disease, and particularly atrophy of mucous membranes, in cases subject to mental disease. Gastric dilation and atrophy of the stomach will occur in a fairly large number of cases And, back of the mere disorder of the membranes, is a striking deficiency in the lymphatic apparatus.[2] This has been studied more especially by Dr. H. M. Adler during his service as pathologist to the Danvers State Hospital. An index to this lymphatic deficiency is afforded by the small size of the spleen characteristic of the insane, to which Dr. Adler has given a statistical form.[3]

To test the validity of the above conclusions concerning the kidney, which were obviously dependent on the sometimes fugitive judgments of many different examiners, and to substantiate, if possible, their naked-eye conclusions by microscopic ones, it was determined to study systematically a random series of a hundred cases in which (a) the same standards of gross examination, (b) the same microscopic technic, (c) the same standards of microscopic examination, (d) the same general source of material, and (e) a single aim in the general logical process of diagnosis, should conspire together to produce reliable conclusions.

The cases specially studied histologically were from the laboratory of the Boston State Hospital. They represent age and sex distribution fairly:

Cases examined, 100; age above 50, 74; age under 50, 26; males, 50; females, 50; females who had borne children, 26.

The ages of the insane at death, so far as they concern the usual autopsy series of a state institution for the insane, are higher on the average than those of general hospital patients. This fact has a bearing on the incidence and nature of renal lesions as they are found in subjects autopsied in state hospitals.

The following shows the age distribution of our cases:

Age 26–30, 2 cases; 31–35, 3; 36–40, 5; 41–45, 4; 46–50, 4; 51–55, 8; 56–60, 6; 61–65, 3; 66–70, 2; 71–75, 2; 76–80, 6; 81–90, 2.

Our series was properly representative of the different disease groups, though it may perhaps be thought that the series is unduly "organic" with respect to the high incidence of coarse brain lesion cases. Careful studies in the somatology of the insane always yield, we think, some such percentages as the following:

Classification according to mental disease:

General paresis and brain syphilis, 30; senile psychoses, 26; arteriosclerotic mental disease, 12; dementia precox, etc., 8; alcoholic psychoses, 6; manic-depressive (including involution melancholia), 6; toxic-exhaustive psychoses, 5; epilepsy and imbecility, 5; unclassified, 2; total, 100. "Organic," 73; non-"organic," 27.

As the interest of our high percentage of renal changes depends somewhat on the findings in cases under fifty years of age, the following is presented. Some pathologists assert that most persons over fifty have traces of renal disease; whether this is or should be the case is a question deserving intensive study by modern methods:

Under fifty years of age:

General paresis and brain syphilis, 14; dementia precox, 5; alcoholic psychoses, 3; toxic-exhaustive psychoses, 2; unclassified, 2.

In order to compare our series of one hundred cases with the larger series above mentioned and to permit other pathologists to compare their naked-eye results readily with our own, we cast into a table the gross findings in the present series:

ANALYSIS OF KIDNEY LESIONS IN 100 CASES.

Chronic lesions.

Chronic lesions in the gross	55
Chronic interstitial nephritis	33
Chronic interstitial nephritis, combined	9
Total gross interstitial nephritis	42
Old infarcts	2
Cystic kidneys	1
Contracted kidneys	1
Fatty kidneys	3
Passive congestion	8
Renal atrophy	1
Chronic diffuse nephritis	3
Renal lithiasis	2
Hydronephrosis, pyonephrosis, pyelonephritis, pyelitis	7

Acute lesions.

Cases examined	100
Acute lesions in the gross	28
Acute parenchymatous nephritis	17
Cloudy swelling	1
Injection	4
Acute congestion	6
	28

It is proper to inquire what these patients in life might have had to show for these lesions. Although this is no part of our present inquiry, we give in the following summary a statement of what could be gleaned from the clinical records of the hospital:

Gross examined anatomically, 100; record of routine urine examination, 65; albuminuria, 25 (38 per cent); cylindruria, 18 (28 per cent); specific gravity below 1015, 18 (28 per cent).

Other clinical features: Edema, 10; seizures or convulsions (of whatever nature), 19; signs of heart disease, 23.

Blood pressure was taken too infrequently in this series, accruing as it did from many years of admissions (although all recently autopsied), and no statistical data of value are available.

As to the possibility that arteriosclerosis might account for much of the high proportion of the disease, it is proper

to say that there was a varying amount of gross generalized arteriosclerosis in seventy-one cases of the one hundred, although the term "generalized" hardly suggests the varying amount in the different organs. In but one case was renal gross arteriosclerosis so striking in the absence of marked gross arteriosclerosis elsewhere as to determine the diagnosis. On the other hand, microscopical vascular changes were found in eighty-seven cases. Fifty-four cases showed arteriosclerosis markedly in a number of other organs, although there was little or no gross renal arteriosclerosis. Forty-two cases showed well-marked intracranial vascular changes.

Chronic interstitial nephritis proved of particular interest. There were thirty-three cases of well-marked coarse change of this sort.

An extensive tabulation (not here presented) was made covering all findings in the gross and microscopically in all the various recognizable structures of the kidney. In the summary of this paper the more important conclusions have been placed, with special mention of (a) plasma cells and their distribution, (b) casts and their distribution, (c) special conditions in general paresis, (d) the tubule of election for cast-retention, (e) a possible seasonal distribution for cast-deposit in one group of cases, etc.

Attention should be drawn to the fact that, although very careful examination of sections prepared by current methods for demonstrating renal structure (eosin and methylene blue and anilin blue connective tissue stains), yet often no more than two or three sections were examined from a given case. We wish to emphasize that these findings do not represent a finical search for lesions rare in a given case. Much more could doubtless have been obtained by examining many more sections from each case; to this we did not proceed because the findings were so rich with our more limited search. Much of interest likewise could have been obtained by a systematic study with methods for demonstrating fat in the tubule walls, as an examination of a number of cases readily showed. But our work was limited to securing a general

statistical conception of the extent to which acute and chronic renal changes are likely to be found in the insane.

In the remainder of the text we place certain facts of special groups of cases.

Plasma cells were found in forty-two cases but never in such quantity as to suggest the acute interstitial nephritis studied by Councilman. Thirty-eight of these cases showed plasma cells about the glomeruli, often in association with a glomerular lesion.

Exceptions were the following:

Cases showing plasma cells *not* about glomeruli but in other loci:

11.25, F., 69, dementia precox, perivascular; 12.49, F., 52, general paresis, subcapsular; 13.4, F., 83, senile psychosis, pelvic; 13.9, M., 85, unclassified, around proximal tubules.

Plasma cells occurred in the perivascular spaces in three cases:

One, 11.25, F., 69, dementia precox, showed none elsewhere. There was generalized arteriosclerosis, cardiac hypertrophy, chronic passive congestion of liver, cholelithiasis, and focal cerebral gliosis. The heart's blood yielded Bacillus Zopfi (usual habitat, intestines of fowls) and the cerebrospinal fluid was negative.

Two cases showed perivascular plasma cells in addition to those found about the glomeruli:

11.8, F., 48, general paresis: The case showed slight general arteriosclerosis, cardiac hypertrophy, chronic myocarditis, a questionable syphilitic interstitial hepatitis, cerebral atrophy and gliosis. The heart's blood yielded Sarcina pulmonium (a non-pathogenic organism usually found in air passages of man); the cerebrospinal fluid yielded Micrococcus pyogenes aureus; bronchial lymph node, negative; decubitus; Bacillus coli.

13 17, F., 83, senile psychosis: In this case the periglomerular plasma cells lay chiefly about those glomeruli just beneath the capsule. The case showed gangrene of lung, but no intra-abdominal lesion, except injection of intestinal and bladder mucosa. There was hydronephrosis, for which no cause could be determined. Acute vegetative endocarditis (Streptothrix invulnerabilis) presumably followed the pulmonary gangrene. The cerebrospinal fluid yielded Planococcus citreus (habitat, tuberculous cavities), B. intestinalis (habitat, excreta of horse), B. fissuratus (habitat, soil), pituitary body (post-pituitary abscess) Micrococcus pyogenes aureus and B. intestinalis. Marked general arteriosclerosis

The following cases showed plasma cells both about the glomeruli and in other loci:

11.8, F., 48, general paresis, perivascular; 11.9, M., 73, senile dementia, general; 13 8, M., 40, taboparesis, pelvic; 13.17, F., 83, senile psychosis, perivascular (the periglomerular plasma cells are subcapsular).

Plasma cells occurred in the intertubular tissue under the pelvic epithelium in

13.4, F., 83, senile psychosis: There was an acute cystitis in this case, as well as acute endometritis and multiple decubitus.

13.8, M., 40, taboparesis: This case showed renal calculus, hemorrhagic pyelitis, ureteritis, and acute hemorrhagic cystitis.

For further statements about plasma cells, the reader is referred to paragraphs 17 to 24 of the conclusions. For statements concerning the distribution of casts, the reader is referred to paragraphs 25 to 27 of the conclusions.

The following shows the distribution of casts in different types of tubule:

Casts in some type of tubule, 73; casts in descending loops of Henle (D.H.), 63; casts in ascending loops of Henle (A.H.), 16; casts in proximal convoluted tubules (P.C.), 12.

(But compare remarks on the occurrence of ten of these in one season of four months.)

Casts in collecting tubules (C.), 11; casts in distal convoluted tubules (D.C.), 5; casts in four types of tubule (see special table), 2; casts in three types of tubule (special table), 7; casts in two types of tubule, 14: (P.C., D.H., 6; D H., A.H., 5; D.H., C., 3); casts in one type of tubule, 50: (D.H., 40; P.C., 4; D.C., 3; A H., 2; C., 1).

There were sixty-three of the seventy-three cast-bearing cases which showed casts in the descending loops of Henle. Forty of these showed casts in no other locus.

Cases with casts in three or more types of tubule:

11.16, M., 57: Taboparetic; D.H., A.H., D.C.; chronic interstitial, plasma cells. 11.47, F., 46: Unclassified; D.H., A.H., C.; chronic interstitial, congestion, cloudy swelling. 12.12, F., 69: Senile dementia, arteriosclerosis; D.H., A.H., C.; chronic interstitial, arteriosclerosis, congestion. 12.19, M., 53: Involution melancholia; D.H., A.H., D.C.; chronic interstitial, arteriosclerosis, congestion. 12.44, M., 76: Senile

psychosis; P.C., A.N., D.H.; chronic interstitial, plasma cells, acute parenchymatous. 13.1, M., 43: General paresis; P.C., A.H., D.H., C.; cystic kidneys, chronic interstitial arteriosclerosis. 13 15, F., 52: Presenile (paranoic); A.H., D.H., C.; cystic kidneys, chronic interstitial, arteriosclerosis. 13 20, M., 65: General paresis; A.H., D.H., C.; chronic interstitial, plasma cells. 13 26, F., 38: Alcoholic hallucinosis; A.H., D.H., C.; chronic interstitial, plasma cells.

There are twelve instances in which casts were found in the proximal convoluted tubules. With the exception of 11.14, 12.14, and 13.25 these cases occurred between numbers 12.44 (autopsied October 12, 1912), and 13.10 (autopsied February 8, 1913), inclusive. These occurred accordingly within a period of about four months. Ten autopsies out of twenty-five (forty per cent) during this period showed casts in this locus.

In two of the cases which did not fall in the period in question there were casts also in the descending loops of Henle.

Three of the cases with casts in the proximal convoluted tubules (series of ten above mentioned) failed to show casts elsewhere.

Sixty-six cases showed more or less glomerular change involving the tuft itself, but in two of the instances the change was of the nature of an acute glomerular nephritis. In thirteen others the change could be described as incipient or as occurring in a few tufts only, so that the total of cases with serious glomerular change of a chronic nature may be set at fifty-one.

Changes affecting the structure of the glomerular capsule are far fewer in number. Twenty-seven cases showed such and ten of these showed changes either slight or infrequent, so that seventeen cases only showed a serious change. Only five cases showed capsular changes in the absence of tuft changes.

Casts were found in eight cases under fifty years of age without tuft or capsule change of any extent. We suppose that the conditions of cast-formation have no necessary relation to glomerular changes.

This latter conclusion and some others are shown below.

For their possible comparative value to other workers we give our findings in cases dying at fifty years of age or less. The tubules in which casts were found are indicated by initials.

CASES DYING UNDER 50 YEARS OF AGE.

	Tuft Capsules Thickened.	Fibrous Tufts.	Plasma Cells.	Casts.
11.7	Some.	Some.	O	D.C.*
11.8	O	Some.	+	D.H.
11.11	O	O	O	O
11.13	O	Slight.	+	O
11.14	Some.	Some (some colloid).	+	P.C., D.H.
11.28	. . . . .	One focus.	+	D.H.
11.30	O	Amyloid.	O	D.H.
11.47	+	O	O	D.H., A.H., C.
12.5	+	Some.	O	D.H.
12.7	O	O	O	A.H.
12.9	Slight.	Some.	O	D.H.
12.14	O	Some.	O	D.H.
12.17	O	Some.	O	D.H.
12.42	Few.	O	O	D.H., A.H.
12.47	O	O	O	P.C., D.H.
12.48	O	O	O	P.C.
12.54	+ Slight.	O	+	D.H.
13.1	O	Few.	+	P.C., D.H., A.H., C.
13.8	Many.	Many.	+	O
13.12	Few.	Few.	O	D.H.
13.14	Slight.	Many.	O	O
13.19	O	Some.	+	D.H.
13.23	O	Some.	+	O
13.26	O	Some.	+	D.H., A.H., C.
13.30	O	O	+	D.H.
13.33	Some.	Some.	O	D.H., A.H.

* For abbreviations see page 292.

Five subjects, fifty years old or less, failed to show casts in any type of tubule. One showed casts in four types (13.1, general paretic), two (11 47, unclassified, and 13.26, alcoholic hallucinosis) showed casts in three types, three in two types, and the remainder (14 cases) showed casts in but one type of tubule.

Some facts about the acute cases are presented in the following table:

Number.	Nature.	Blood Culture.	Infective Focus in Body.	Infective Focus in Genito-urinary Tract.
1911.3.	Cloudy swelling.	O	Acute vegetative endocarditis.	Chronic cystitis.
1911.4.	Acute parenchymatous nephritis.	+*	Bronchopneumonia.	
1911.7.	Acute parenchymatous nephritis.	O	Acute fibrinous pericarditis.	
1911.10.	Injection.	O	Purulent otitis media.	Acute cystitis.
1911.11.	Acute diffuse nephritis.	+	" " "	
1911.13.	Injection.	O	Pulmonary tuberculosis.	
1911.24.	Acute parenchymatous nephritis.	O	Bronchopneumonia.	
1911.28.	Acute congestion.	+	"	
1911.29.	Injection.	+	Decubitus. Acute gastric ulcer.	
1911.30.	Acute congestion.	O	Perforated esophagus and stomach.	
1911.33.	" "	O	Fibrinous pleuritis.	" "
1911.38.	Acute parenchymatous.	O	Acute ulcerative colitis.	" "
1912.1.	" "	+	Bronchopneumonia.	Chronic cystitis
1912.3.	" "	+	Acute vegetative endocarditis.	Acute cystitis.
1912.12.	Injection.	O	Lobar pneumonia.	" "
1912.13.	Acute parenchymatous.	O	Purulent otitis media — liver abscess — bronchopneumonia.	
1912.14.	Acute diffuse nephritis.	O	Purulent otitis media.	
1912.15.	" " "	O	" " "	
1912.17.	Acute parenchymatous.	O	Bronchopneumonia.	
1912.43.	Injection.	+	"	" "
1912.48.	Acute parenchymatous.	+	"	
1912.49.	" "	+	Pyelonephritis.	" "
1912.58.	Acute congestion.	O	Surgical wound right elbow.	" "
1913.1.	Acute parenchymatous.	O	Tuberculous ulcers ileum.	Chronic cystitis.
1913.3.	" "	+	Cellulitis face.	
1913.12.	" "	+	Decubitus.	
1913.14.	" "	+	Submucous hemorrhages.	Acute cystitis.
1913.16.	" "	+	Gangrene of lung.	Chronic cystitis.

* + indicates that bacteria were grown.

TABLE. — *Continued.*

Number.	Nature.	Blood Culture.	Infective Focus in Body.	Infective Focus in Genito-urinary Tract.
1913.20.	Acute parenchymatous.	O	Diphtheritic colitis.	Acute cystitis.
1913.23.	" "	+	Bronchopneumonia.	
1913.24.	" "	+	Cellulitis scalp. Pyopericardium.	
1913.30.	Acute congestion.	+	Pyelitis.	" "
1913.31.	Acute parenchymatous.	+	Acute aortic vegetation and pyelonephritis.	Chronic cystitis.
1913.34.	" "	+	Fibrinous pleuritis.	
1910.15.	" "	O	Pyelitis — putrid bronchitis — abscess intestinal wall.	" "
1910.16.	" "	O	Acute fibrinous pleuritis.	

CONCLUSIONS.

1. These general results substantiate those of a more superficial inquiry in a larger number of cases (see introductory note) as well as those of the late W. L. Worcester on the same kind of material (1899).[4]

2. The inquiry here reported deals with a more systematic histological examination of kidneys in the insane than has been reported for many years, embodying a tabulation of findings in the gross and microscopically in the different recognizable structures of the kidney.

3. The analysis permits saying that normal kidneys must be of the greatest rarity in the insane at autopsy, for in the present series of one hundred no instance of normal kidneys was found.

4 It is less possible to say that these renal conditions were of moment to the individuals who bore them, since some of the lesions are very possibly extinct and others cannot safely be interpreted in the present state of pathology.

5. Their interest from the therapeutic and dietetic standpoint is considerable, since there were at least thirty-nine instances of acute renal disease and eleven of these complicated by a background of chronic lesions.

6. Aside from these thirty-nine acute (or acute and chronic) conditions, there were fifty-five instances of chronic lesions (or sixty-six, if we include the eleven cases with both acute and chronic lesions).

7. There were in point of fact but five cases in which the kidneys were regarded as normal to the naked eye.

8. Clinically, among sixty-five cases examined, albuminuria was found in twenty-five (or thirty-eight per cent), and cylindruria in eighteen (or twenty-eight per cent). Also the specific gravity went below 1015 at times in eighteen cases (or twenty-eight per cent). No special statistical significance need be attached to these latter figures.

9. Clinically also there were ten instances of edema, probably either caused or favored by the renal condition (four of these ten cases showed cardiac disease also).

10. Clinical records show nineteen instances of seizures or convulsions of some sort, but it is not clear how many of these can be regarded as renal (see note below concerning general paresis).

11. Clinical records also indicate that twenty-three of the hundred cases were regarded in life as more or less severe cardiac cases.

12. Of the females examined, twenty-six (or fifty-two per cent) had borne one or more children.

13. Thirty-two of the one hundred cases were emaciated at death.

14. The most prominent gross lesion in the series was chronic interstitial nephritis, which occurred in forty-two cases.

15. Microscopically, chronic interstitial nephritis was found not only in these forty-two cases, but also in twenty-four other cases (a total percentage of sixty-six).

16. There were thirty-three cases in which chronic interstitial nephritis was not only marked in the gross but was the only significant kidney finding microscopically also.

17. Microscopically, much attention was paid to the occurrence and distribution of plasma cells in the kidney substance, since these might well be regarded as indicating a more active (or less extinct) sort of lesion than simple fibrosis.

18. Plasma cells were never found in such quantity as to suggest acute interstitial nephritis. But plasma cells were found in forty-two per cent of the series.

19. Thirty-eight of these forty-two plasma cell cases showed the plasma cells distributed chiefly about the glomeruli. This distribution naturally suggests special conditions (toxic ?) in the periglomerular region, and in point of fact there was very frequently a glomerular lesion associated with this exudation.

20. A broader or different distribution of plasma cells was far less common, and the small groups of such unusual distribution are presented in the text.

21. Eleven cases out of twenty-six under fifty years of age yielded plasma cells, *i.e.*, exactly the same percentage as did the total series.

22. The occurrence of plasma cells in the kidneys of general paretics is worthy of note by reason of their constant occurrence in the brains. Seventeen of thirty paretics in this series showed plasma cells in the kidney, or fifty-six per cent. It is possible that they are of focal occurrence in the kidney (though nothing specially to indicate this was found), and that more systematic work would swell the percentage. Sixteen of the seventeen paretics showed the plasma cells in the periglomerular region while one showed them in a subcapsular zone.

23. Seven of the whole series of thirty paretics had seizures; five of these seizure cases showed periglomerular plasma cells, one showed no chronic lesion except general fibrosis, and one was a case of acute parenchymatous nephritis without chronic lesion (12.13). But three of these seven cases showed casts, and these only in one tubule type (descending loop of Henle).

24. Curiously enough, twelve other non-paretic cases which had seizures or convulsions of various sorts failed to show plasma cells in the kidneys, but in eight instances did show casts, although always confined to a single tubule type (six times in the tubule type of election, the descending loop of Henle).

25. Seventy-three cases showed casts in one or more types of tubule; fifty cases in one type of tubule only (forty, descending loop of Henle); fourteen in two tubule types; seven in three tubule types; and two in four tubule types.

26. Sixty-three of the seventy-three cast-bearing cases showed the casts in the descending loop of Henle, which seems entitled by consequence to be called the tubule of election for cast deposit or retention.

27. There was a curious time distribution of those cases which showed casts in the less common locus of the proximal convoluted tubule. Ten of these twelve cases died within a period of four months, November to February, 1912–1913. One may suspect special dietary or bacterial conditions for this fact.

28. Glomerular tuft changes of a serious nature occurred in fifty-one cases and there were indications of disease in thirteen others (besides two acute lesions). Changes in the glomerular capsule were far less in number, being of a serious nature in but seventeen cases (slight or infrequent in ten others). Cast deposits occurred in eight cases under fifty years of age without evidence of glomerular change.

29. Some facts are noted concerning the possible relations of acute renal lesions to infective foci in the urinary apparatus or in the body at large.

30. The study seems to show a significantly high proportion of chronic and acute lesions of the kidney in psychopathic subjects; such conditions should engage the attention of dieticians in insane hospitals.

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XLI

PROGRESS OF THE PSYCHOPATHIC HOSPITAL ON THE PROPHYLACTIC SIDE OF MENTAL HYGIENE.*

By E. E. Southard, M.D., Boston,

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I wish to call attention today to but three components of our recent progress on the prophylactic side of mental hygiene. The fourth component, viz., the out-patient department, with its after-care technic, I leave to other speakers. Nor shall I deal with these three components, viz., (*a*) the so-called temporary care service of the Psychopathic Hospital; (*b*) our voluntary admissions, and (*c*) the admissions which prove to be "not insane," with any attempt at completeness. These topics will, in point of fact, be treated more comprehensively in a paper to be published in the *Journal of the American Medical Association* embodying statements made before the Nervous and Mental Section of the American Medical Association at the Atlantic City meeting (1914), where was held a symposium on the prevention of insanity. That symposium was held yesterday morning and provoked a lively discussion. Resolutions were forwarded by the section asking that the American

* Being Contributions from the Psychopathic Hospital, Boston, Number 52 (1914.18) read in abstract at the Second Annual Conference on the Medical and Social Work of the Psychopathic Hospital, Boston, Mass., June 26, 1914. (*Bibliographical Note.*—The previous contribution, Number 51 (1914.17), was by Walter Channing, entitled "The Duty of the State to the Psychopathic Hospital," published in the Boston Medical and Surgical Journal, Vol. clxxi, No. 23, December 3, 1914.

Medical Association's Council on Public Health and Instruction be requested to take up this matter as a recognized constituent of its otherwise most comprehensive program.

The advantage which the mental hygiene movement is now showing is the advantage of concrete proposals backed by increasing statistical evidence. As Mr. Clifford W. Beers is fond of saying, "It is difficult to keep the mental hygiene propaganda from 'turning into a letterhead.'" I believe that the beginnings of progress which the Psychopathic Hospital here in Boston has made will go far to scotch the letterhead tendency. What I shall now try to do is to state as briefly as possible the main conclusions of the longer paper read before the Section of Nervous and Mental Disease of the American Medical Association.

A. The first great advance in the prophylaxis of mental disease in which the Psychopathic Hospital may be said to have taken a leading part, is an advance of which Massachusetts alone can, I believe, at present boast, since it depends upon the remarkable law giving temporary care to persons suffering from mental derangement which was passed in 1911 (Acts of 1911, Chapter 395).† I do not need to rehearse the law in

† An Act Relative to the Reception and Temporary Care in Certain Institutions of Persons Suffering from Mental Derangement.

Be it enacted, etc., as follows:

The superintendent or manager of any hospital for the insane, public or private, may, when requested by a physician, by a member of the board of health or a police officer of a city or town, by an agent of the institutions' registration department of the city of Boston, or by a member of the district police, receive and care for in such hospital as a patient, for a period not exceeding seven days, any person who needs immediate care and treatment because of mental derangement other than delirium tremens or drunkenness. Such request for admission of a patient shall be put in writing and filed at the hospital at the time of his reception, or within twenty-four hours thereafter, together with a statement in a form prescribed or approved by the state board of insanity, giving such information as said board may deem appropriate. Such patient who is deemed by the superintendent or manager not suitable for such

detail and will merely mention that as all physicians and social workers now know, it is possible for any physician, a member of the board of health, or police officer in a city or town, an agent of the Institutions Registration Department of the City of Boston, or member of the district police, to request the superintendent or manager of any hospital for the insane to receive for a period of seven days and care for any person needing immediate care and treatment because of mental derangement. Under this law, the old judicial procedure is not employed. I must emphasize that the law is not open to the charge that the individual liberty of the prospective patient is interfered with to any extent; for, although various persons who may request such care for a prospective patient may not be experts in mental disease, yet it remains optional with the superintendent or manager of the hospital whether he shall receive or not receive the person in question as a patient. The law has been of untold benefit to hundreds upon hundreds of patients.

I am tempted to think that the passing of such a law and the establishment of hospital facilities with which to operate the law are measures of

care shall, upon the request of the superintendent or manager, be removed forthwith from the hospital by the person requesting his reception, and, if he is not so removed, such person shall be liable for all reasonable expenses incurred under the provisions of this act on account of the patient which may be recovered by the hospital in an action of contract. The superintendent or manager shall cause every such patient either to be examined by two physicians, qualified as provided in section thirty-two of chapter five hundred and four of the acts of the year nineteen hundred and nine, who shall cause application to be made for his admission or commitment to such hospital or, provided he does not sign a request to remain under the provisions of section forty-five of said chapter five hundred and four, to be removed therefrom before the expiration of said period of seven days. Reasonable expenses incurred for the examination of the patient and his tranportation to the hospital shall be allowed, certified and paid as provided by section forty-nine of said chapter five hundred and four, as amended by chapter four hundred and twenty of the acts of the year nineteen hundred and ten, for the allowance, certification and payment of the expenses of examination and commitment.

primary importance in the mental hygiene movement so far as that movement can be mechanized by public institutions. Parenthetically, I would say that I feel that the success even of privately endowed mental hygienic measures (out-patient departments, reference bureaus and the like) will be in the long run dependent upon having in the background properly constituted public institutions to which certain cases may be referred.

Of course such temporary care cases may be admitted to all hospitals for the insane and the passing of such laws greatly stimulates state hospitals to provide proper facilities for the reception of these patients. These facilities may be briefly stated as the facilities of a general hospital plus those of a hospital for the insane. These are precisely the facilities which the Psychopathic Hospital in Boston has to offer, with arrangements for research added thereto and the whole greatly aided by the out-patient department and the extramural activities of the social workers connected therewith.

After the passage of the temporary care law (Chapter 395, Acts of 1911) there came to Massachusetts institutions 92 persons under the act during 1911. These admissions were then approximately at the rate of 200 per annum. This rate was impressively increased during 1912 to 416; the opening of the Psychopathic Hospital upon June 24, 1912, was of course, a tremendous stimulus to this increase and for this year we have to report the almost astounding figure of 897 such admissions. Five hundred and ninety of these 897 admissions during 1913 were to the Psychopathic Hospital alone, the other 309 to other state institutions. As to the advantage of passing a patient through this seven days' period

of temporary care, I need not elaborate the obvious, except to say that whether or not the patient is committed or discharged, he must possess the feeling that he has been given a fair chance to prove the suspicion of mental derangement (entertained by his relatives or by experts) to be right or wrong.

B. Perhaps it would be difficult to convince the law-makers of less civilized communities that the judicial procedure ordinarily in vogue could be safely suspended and replaced by a law like our temporary care law in Massachusetts, but, at all events, our experience with respect to voluntary admissions is so voluminous and cogent, that certainly no legislature of any state, not now possessing laws concerning the voluntary admission of insane persons to public hospitals, could fail to be convinced of the value of some such voluntary admission law. There are, however, many states in the Union which do not possess such laws, and many of the states which do possess them do not permit the public support of voluntary patients, so that the operation of the law is not extensive enough to prove of great value to the community. The law in Massachusetts has in fact been upon the statute books since 1881, but there was no especial stimulus to the voluntary admissions until the year 1905 when the State Board of Insanity's interest in this matter caused the law to be amended so as to permit public support of voluntary admissions.* Thereafter the admissions rose grad-

* Section 45. The superintendent or manager of any institution, public or private, to which an insane person may be committed, may receive and detain therein as a boarder and patient any person who is desirous of submitting himself to treatment, and who makes written application therefor and whose mental condition is such as to render him competent to make the application. Such person shall not be detained for more than three days after having given notice in writing of his intention or desire to leave the institution. The charges for the support of such person in a state institution

ually from 5% of the total intake (such intake being defined as composed of the regular commitments plus the voluntaries) in 1906 to 8% in 1912. The figures were not so impressive numerically during those years as they are impressive from the percentile standpoint, in view of the fact that in 1906 there were only 125 such voluntary admissions and in 1912 only 282. Now, however, we have to report the astonishing fact that during 1913 there were 636 persons who were either admitted under the voluntary law or became voluntary patients later. This amounts to 16% of the total intake for 1913, which was 4051 patients in Massachusetts. As a whole, this leap from 8 to 16% voluntary admissions within a single year is indicative of what the establishment of proper facilities in a community means. I have no doubt that it is safe to assert that the large increase has been greatly determined both by the public effect which the Psychopathic Hospital in Boston has exerted and by the endeavor which the Psychopathic Hospital officers have had fully in mind to get all patients registered under the voluntary law so far as possible. Yet I would not leave you with the impression that we have registered persons under the voluntary law who were unable to understand their status as such. In point of fact, the State Board of Insanity has rightfully been very jealous lest persons should be registered as voluntary patients when they did not understand their status. Accordingly, if a patient does not understand his right to secure discharge from the institution within three days of his application in writing for such discharge, the authorities take steps to have the patient's status changed.

shall be governed by the provisions of law applicable to the support of an insane person in such institution, provided the approval of the state board of insanity shall be obtained in writing.

Of course certain voluntary patients pass through intervals of confusion or excitement or deep depression during which it may be difficult to elicit statements from them concerning their status. Here a certain amount of latitude may well be allowed in the curable group of patients. Nor do I understand that the Board of Insanity desires us to change the voluntary status in such episodic cases.

The extent to which the temporary care law and the voluntary admission law are reducing the court commitments may be stated as follows, on the basis of the Psychopathic Hospital admissions for 1913. There were 1391 admissions which were not under the regular court commitment law during 1913. Most of these were under the temporary care or voluntary admission laws, although there were 394 under the Boston police law, Chapter 307, Acts of 1910, a law resembling in many respects the temporary care law. Into the special features of Chapter 307, I do not care to go in this place, since the law which we would like to see enacted by other states is rather an improved temporary care law (Chapter 395, Acts of 1911) than an improved Boston police law (Chapter 307, Acts of 1910). The point I wish to make here is that of the 1391 admissions under various special laws (exclusive of all regular court commitments) 665 were for various reasons spared the process of regular court commitment, that is to say, almost one-half, or 48%, of the total intake under various special laws were spared the stigma and expense of such commitment.

C. With this brief statement, I will dismiss the matter of temporary care and of voluntary admissions, and will proceed to the matter of the "not insane" discharges at the Psychopathic

Hospital. I think it will astonish even the specialists in this field to learn that of the first 2500 discharges from the Psychopathic Hospital, involving a period from the opening of the hospital, June 24, 1912, to March 20, 1914, no fewer than 570 cases were discharged as "not insane." This figure warrants our saying that the Psychopathic Hospital in Boston, operating under certain laws favorable thereto, almost deserves the term "mental and nervous clinic." In Germany, many institutions resembling in scope the Psychopathic Hospital in Boston, receive the name "Psychiatrische und Nervenklinik." We had not hoped so soon in Boston to arrive at such a combination of psychiatric and neurological interests. I will not go into the diagnoses of these 570 cases at all in detail at this time, but will call attention to the fact that no fewer than 179 of these cases were determined to be mentally deficient, and that perhaps one-third or more of these mentally deficient cases were delinquent or potentially delinquent persons. The result of this situation is that, beyond question, the Psychopathic Hospital can aid in the solution of some of the problems of criminology, if we are willing to admit that the alienist has anything to contribute in the direction of criminology. This, I believe, we must admit, both on general grounds and on the special ground of recent work, of which I need only mention that of Dr. William Healy at the Juvenile Psychopathic Institute in Chicago. Nor will I mention in detail the somatic as well as more strictly neurological cases that for one reason or other came to the Psychopathic Hospital and were discharged as "not insane." I wish to speak more emphatically at this time of the 100 cases belonging in the group of psychoneuroses. The

question for us to decide is, *should there be a public institution having the function of a preventorium to receive these psychoneurotic patients?* Further experience will be necessary before we can assert on statistical grounds that such a preventorium is necessary, although I am willing to go so far as to say that, in my opinion at the present time, the number of convalescent mental cases is almost sufficient, taken in conjunction with the psychoneurotics, to warrant the establishment of such a hospital, provided that funds are forthcoming. The question, it is true, was answered in the negative some years ago by the commission appointed by Governor Draper to inquire into this matter. However, at that time, the commissioners had not the experience of an institution like the Psychopathic Hospital to draw from. It must be then one of the concrete tasks of the immediate few years to determine the desirability of such a preventorium and convalescent home and to work out the proper features and best site of such a hospital.

What we can safely say at the present time is that an institution like the Psychopathic Hospital in Boston is no deterrent to the resort of psychoneurotics. In many of these cases there is evidence such as legitimately to warrant the suspicion of their mental derangement. The operation of the temporary care law, of the voluntary admission law, and even to some extent of the Boston police law (Chapter 307, Acts of 1910) has been to tap society in a new stratum which I believe has not hitherto been tapped to any extent by public institutions; but to operate these laws most effectively an institution like the Psychopathic Hospital is necessary. Massachusetts' experience should be utilized by other

states in the direction of the enacting of such laws and the establishment of such psychopathic hospitals and wards as are now available and operative so successively in our commonwealth. It can conservatively be stated that these measures will go far toward the abolition of the stigma of insanity and the stimulation of earlier resort to public and private institutions. In point of fact, through the operation of various causes, of which the laws mentioned and the Psychopathic Hospital are but symptoms, the stigma of insanity is passing both in the public practice of institutions and in the private practice of physicians. It will not be many years before practitioners known as alienists will have prospective patients flocking to their doors much as such patients now flock to the Psychopathic Hospital, and indeed I find that psychiatrists, formerly masquerading as general practitioners and neurologists, are now more willing to admit that they are really psychiatrists. They do not find that this admission operates to turn prospective patients away. The close atmosphere of the courts is being replaced by the bracing atmosphere of the modern clinic.

I will sum up briefly by saying that the prophylactic division of mental hygiene can safely claim to be far more than a letter head or a propaganda and that, whatever its legal and public institutional sides, the prophylactic division of mental hygiene has as concrete measures:—

1. The stimulation of proper temporary care of persons suffering from mental derangement under the conditions of general hospital and private practice.

2. The stimulation of voluntary admissions to existing and future hospitals for the insane.

3. The establishment of psychopathic hospitals in proper centers, having proper medical and social arrangements for the highest forms of intramural and extramural individual and community service. If you are tempted to state that the term "psychopathic" somewhat resembles that blessed name "Mesopotamia" in its drawing powers, as Mr. Frank B. Sanborn once insisted, yet I venture to hope that its extension to include both the legally insane and the great variety of other mental cases including psychoneurotics, mentally deficients, and criminalistic and possibly other types of mental disorder, will tend to abolish the use of the term "insane" by physicians, except under court conditions. The term "insane" is rightfully considered a legal and not a medical term. One of the greatest features of a mental hygiene propaganda will be to convince and to persuade the world of this fact.

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XLII

THE MARGIN OF ERROR IN PSYCHOPATHIC HOSPITAL DIAGNOSES.*

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and

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The margin of error in the diagnosis of mental disease is a matter of social importance. For upon the medical diagnosis of mental disease hangs the legal decision as to insanity. It is notoriously unsafe to say that a man is insane unless the particular form of mental disease producing his insanity can be specified. To be sure, we often hear that such a man is insane,—but "what kind of insanity" cannot be specified. It is a question, however, whether there are any "kinds" of insanity, at least any kinds corresponding to the forms of mental disease. A man is insane, or he is not insane, or, if there be the so-called demi-insane, the new group

* Being Contributions from the Psychopathic Hospital, Boston, Number 61 (1914.27), read at the Second Annual Conference on the Medical and Social Work of the Psychopathic Hospital, June 26, 1914. (*Bibliographical Note.*—The previous contributions, Number 60 (1914.26), was by Frankwood E. Williams, entitled "Cases to Illustrate Symptomatic Psychoses of Cardiorenal Type," published in Boston Medical and Surgical Journal, Vol. clxxi, No. 24, December 10, 1914.)

either represents an eventual improvement in legal definitions (*e.g.* of grades of responsibility) or else the term demi-insane represents an impossible endeavor to break up the legal term "insanity" into the medical subdivisions of mental disease. Although strictly speaking, the medical man has no more than the ordinary citizen's right to assert insanity, yet practically speaking, the medical man (or as a rule, two medical men speaking as one) has much to say, since his medical diagnosis "mental disease" *may* mean the legal decision "insanity."

If then there are mental diseases from the medical standpoint which are not equivalent to the legal status of insanity, it is clear that the diagnosis of the form of mental disease may rise into a social function of the greatest delicacy. Studies of the error of diagnosis as to form of mental disease should accordingly be made from time to time in properly managed clinics and particularly in those which have the advantage of dealing with numbers of psychopaths (*i.e.* victims of mental disease) that are determined to be not insane.

We accordingly make no apology for the present orientation study of the problems of medical diagnosis at the Psychopathic Hospital, Boston, since it would be difficult to find a clinic more representative of the entire range of psychiatric problems and since a major function of the institution (with its intake running close to 2000 patients a year) is the diagnostic function. Moreover, the time-relations are such, particularly under the provisions of the Massachusetts seven days' "temporary care" law, that rapid diagnosis is a desideratum. Where rapid diagnosis is not merely the usual godsend to the patient, but becomes an official necessity, condi-

tions are particularly favorable to the study of our present day powers in the field of psychiatric diagnosis.

Distributing patients as the Psychopathic Hospital does (under conditions prescribed by the State Board of Insanity), to a number of state institutions, each with its own well-defined traditions in diagnosis, the Psychopathic Hospital has the advantage of having its work controlled by a number of minds.

We should not, however, leave the impression that what we are studying is the accuracy of "snap" diagnoses. The average stay of patients at the Psychopathic Hospital is between two and three weeks, and legal devices are such that in most instances difficult cases can be kept indefinitely for diagnostic or therapeutic purposes. Hence the diagnoses recorded at the hospital are as a rule not snap diagnoses. They are diagnoses made as rapidly as diagnostic devices will permit, under standards intended to be as high as those of the best general and insane hospitals.

Readers who do not know their Massachusetts might believe that the state institutions to which our patients are sent would accede too readily to Psychopathic Hospital diagnoses and hence tend artificially to lower our percentage of error. Those who know the individualistic and fighting spirit of Massachusetts will not fear this diagnostic artifact.

Our point of view in this study is, therefore, to scrutinize the diagnostic results of the Psychopathic Hospital, determine the preventable errors in diagnosis, delimit the region of greatest error, and discover the region in which research should best proceed.

Studies of somewhat similar scope had been

carried out with Danvers State Hospital material, utilizing, however, autopsied cases and the staff-meeting diagnosis as the basis of analysis.

In 1910 it was shown that, *without* the use of the Wassermann reaction, the accuracy in diagnosis of general paresis might be variously stated as from 85% to 90%.

Orton has published a similar study from Worcester, and Dr. Myrtelle M. Canavan has given us similar figures from the Boston State Hospital. These are embodied in the following table:

TABLE SHOWING ERRORS IN DIAGNOSIS OF GENERAL PARESIS WHERE THE WASSERMANN REACTION WAS NOT EMPLOYED.

	Clinical Diagnoses, Paresis.	Histological Not Paresis.	Diagnoses Not Any Form of Neurosyphilis.
Southard, 1910...	41	6	3
Orton, 1913.....	60	9	8
Canavan, 1914 ...	18	3	2
	119	18	13

Thus general paresis was wrongly ascribed to 18 cases in 119, or in 15%. Neurosyphilis (*i.e.* not merely general paresis, but also other neurosyphilis) was wrongly ascribed to 13 cases in 119, or 11%.

	Clinically Not Paresis.	Proved Paresis.
Southard, 1910.....	186	2
Orton, 1913.....	116	1
Canavan, 1914.....	106	1
	408	4

General paresis has already turned up in 4 cases in 408 cases in which the diagnosis of pa-

resis was never even remotely entertained. A more intensive examination of a number of these 408 would probably somewhat increase this percentage of error.

In 1910, also, another Danvers study was published showing a surprisingly low percentage (66%) of accuracy in the diagnosis of senile dementia, counting as correct such diagnosis in cases having either brain atrophy or coarse brain lesions due to arteriosclerosis. The effect of this study was to point out the existence and numerical importance of senile psychoses which were consistent with normality of brain or with merely microscopic or chemical changes not resulting in visible destruction of brain tissue.

From a third Danvers study of 1910 may be reproduced three tables illustrating the general accuracy of state hospital diagnoses.

TABLE I.

	Cases
Cases at daily clinics, 1904-8, later autopsied	250
Clinical diagnoses unanimous	184
Revised on autopsy data	21
Opinions divided or doubtful	66
Every opinion incorrect as shown by autopsy	7.
Remain as yet obscure	10

DIAGNOSTIC ACCURACY.

	Cases
89% with all agreed	184
74% some one correct	66
85% some or all correct	250

TABLE II.

	Cases
Unanimous diagnoses	184
General paresis, clinically	42
General paresis, anatomically	36

General paresis, errors	6
Organic and senile dementia, clinically	80
Brain atrophy or cortical arteriosclerosis	62
Tumor, lues, porencephaly, *general paresis*	4
Total organic, anatomically	66
Errors—senile acute psychoses	14
Total errors, general paresis	6+1= 7
Total errors, senile psychoses	14

TABLE III.

Doubtful or obscure cases		66
One or more diagnosticians right		49
Organic and senile dementia	19	
Acute psychoses	14	
General paresis	9	
Brain tumor	4	
Miscellaneous	3	
No diagnostician right		7
(One case of each general paresis, cortical arteriosclerosis, organic dementia, streptococcus septicemia, epilepsy, streptococcus meningitis, cerebellar abscess.)		
Obscure, both clinically and anatomically		10
Acute psychoses (?)	6	
Alcoholic insanities (?)	2	
Deliria (?)	2	

Similar studies from other state institutions are, so far as we are aware, not available, nor is the present study as accurately controlled as these Danvers and Worcester studies, since we here supplant the autopsy diagnoses with clinical diagnoses. It is upon the background of such a study as this, however, that future controls by extensive autopsy studies can be most effectively made.

In many cases the Psychopathic Hospital officers failed to make a diagnosis, terming the case "unclassified," meaning thereby "psychopathic" in some sense, as a rule also "insane" in the sense of certifiable, but as not capable of further specification. That the officers of the

Psychopathic Hospital should not be unwilling thus to confess ignorance was all the more likely on account of the fact that three of its officers (the writers and the chief-of-staff, Dr. H. M. Adler) had been trained at Danvers Hospital, where the habit of leaving cases "unclassified" had become ingrained. The following table shows this:—

UNDIAGNOSTICATED COMMITMENTS.

	In Massachusetts Hospitals.	In Danvers Hospital.	
1904.....	91	26	(29%)
1905.....	93	34	(37%)
1906.....	58	28	(48%)
1907.....	81	18	(22%)
1908.....	92	39	(42%)
1909.....	93	30	(32%)
1910.....	102	44	(43%)
1911.....	119	50	(42%)
1912.....	137	34	(24%)
1913.....	247	41	(16%)
	703	204	(29%)

That this habit of confessing ignorance has been maintained can be seen from tables published in 1913 report of the Psychopathic Hospital, where are given the following figures:—

TEMPORARY CARE GROUP.

Cases	1022	
Unclassified	247	(24 per cent.)
Males	522	
Unclassified	110	(21 per cent.)
Females 500		
Unclassified	137	(27 per cent.)

Analysis shows that the sex-difference is due to the easy resolution of the many more alcoholic and paretic cases among the males which far more than offsets the larger number of "not insane" women in the temporary care group.

VOLUNTARY GROUP.

Cases	362	
Unclassified	68	(19 per cent.)
Males	186	
Unclassified	32	(17 per cent.)
Females	176	
Unclassified	36	(20 per cent.)

Analysis of the voluntary group shows a similar state of affairs to that in the temporary care group. If we combine these figures we arrive at 315 cases unclassified among 1382 diagnoses, *i.e.* 23%.

Of these "unclassified" mental cases, some are to be later resolved through the development of the disease or the accession of new facts, some will remain unresolved through passing prematurely out of observation, some were actually diagnosticable on available facts, and some are essentially not classifiable in the present stage of psychiatry.

We have studied the fate of a group of "unclassified" cases of 1912-1913 as transferred to the institutions receiving the largest number of our cases.

UNCLASSIFIED CASES AMONG THOSE SENT FROM PSYCHOPATHIC HOSPITAL TO VARIOUS INSTITUTIONS, 1912-1913.

	All Cases Sent.	Unclassified.	Remained Unclassified.	Added Unclassified.	Total Remaining Unclassified.
Boston	305	30	3	13	16
Westboro ..	136	40	2	5	7
Taunton ...	91	20	6	2	8
Worcester .	72	23	2	0	2
Danvers ...	29	7	2	3	5
	633	120	15	23	38

It is somewhat curious that the residuum (38 cases) remaining "unclassified" after review by

two hospitals should represent 6% of the total. It may be remembered that the Danvers cases of "obscure" mental and anatomical diagnosis (later worked up by L. B. Alford) were 10 in number, representing 4% of the total of autopsied cases then reviewed.

The percentage of cases sent unclassified from the Psychopathic Hospital was in this group 19%, somewhat less than the 23% stated above for the official tables for 1913. The two case-groups are not identical, though they overlap.

Let us now approach the special problems of change of diagnosis in the separate institutions; we will dismiss the "unclassified" group (except for incidental mention) and study the differences of opinion which are definite and presumably based upon differences in observation or interpretation (omitting merely nomenclatural differences so far as possible.)

	Cases All Sent.	Altered Diagnosis.	%	Cases Minus Unclassified.	Altered Diagnosis.	%
Boston	305	89	29	275	62	23
Westboro	136	62	46	98	24	24
Taunton	91	34	37	85	28	33
Worcester	72	31	43	53	12	23
Danvers	29	14	45	22	5	23
	633	2300	36	533	131	25

That is to say, omitting the 19 and 23 cases out of every hundred in which the Psychopathic Hospital fails to make a diagnosis and considering only those in which a definite diagnosis is made, we find that in 25 of every such 100 a change in diagnosis is made.

The later diagnoses have every chance of being more nearly correct than the earlier, but possibly they should not stand so much chance of being better, as these figures might indicate.

Special analyses for the above five institutions may be made as follows:—

The group of cases sent from the Psychopathic Hospital to the main wards of the Boston State Hospital is an important group by virtue of its size and frequent opportunities of interchange of views by members of the Psychopathic and main staffs.

There is a superficial discrepancy of opinion as to 89 in 305 cases (1913), *i.e.* 29%. In 27 of these 89 cases the psychopathic staff was unable to render an opinion other than "unclassified," by which term some form of insanity is meant. These are failures to diagnose rather than errors, so that true discrepancy of opinion is found in 62 cases in 278 where both staffs arrived at some conclusion, that is, in 22%. In 7 cases one of two Psychopathic Hospital diagnoses was pitched on as correct, so that absolute error cannot be laid at the door of the Psychopathic Hospital. This leaves 55 cases to be considered.

A striking feature of this remainder is that the Psychopathic Hospital diagnosis of manic-depressive insanity is practically never changed (there was one instance, 11589, regarded as infective-exhaustive rather than manic-depressive at the main hospital. The case was one of mania in the course of a pneumonia, and perhaps should be called a symptomatic psychosis, pneumonic type).

On the other hand, the Psychopathic Hospital diagnosis of dementia precox was altered by the main hospital to manic-depressive insanity in some 16 instances. Here possibly there is evidence of two different points of view.

One hundred and thirty-six cases were transferred from the Psychopathic Hospital to the Westborough State Hospital in 1913. In 62

of these (46%) a change of diagnosis was made, but in 38 of these, the change was merely from "unclassified" to a definite diagnosis. Actual differences, therefore, were registered in 24 out of 98 cases (24). But, as in 5 of these the diagnosis became again "unclassified" we are left with 19 cases in which definite diagnoses were rendered by both staffs and disagreement ensued (20%).

Among these 19 cases of disagreement there were five which are of special interest, being alterations from manic-depressive insanity to dementia precox (v. below). There was but one case of the converse change—from dementia precox to manic-depressive insanity (10974)—a fact rather surprising at first blush when the changes of diagnosis at Boston Hospital are considered. Dementia precox was altered to hysteria (10736,—10810), to alcoholic (10938), to imbecile (10972), and to not-insane (10720). Besides the five alterations from manic-depressive insanity to dementia precox, there were four other alterations in the same direction (10625 from general paresis, 10930 from alcoholic, 10,950 from senile dementia [new facts!], 11033 from imbecility). In this connection it is interesting to note that 13 unclassified cases had diagnoses altered to dementia precox, as against 7 unclassified to manic-depressive insanity. Two cases of supposed alcoholic insanity were determined to be manic-depressive (10800, 10824); 10835 became from senile dementia a case of Korsakoff's psychosis; 10893 was altered from organic to senile dementia.

Of 91 cases transferred to Taunton State Hospital, 34 yielded alterations in diagnosis (37%). But 6 of these were from "unclassified," so that the more definite error may be set at 28 in 85 cases (38%). There are four instances in the

Taunton review of alterations of our diagnosis of manic-depressive psychosis to dementia precox.

Seventy-two cases were transferred from the Psychopathic Hospital to the Worcester State Hospital in 1913. In 31 of these (or 43%), a change of diagnosis was made, but in 19 of these the change was merely from "unclassified" to a definite diagnosis. Actual differences of opinion, therefore, were registered in 12 out of 53 cases (or 23%). There were no instances in this Worcester Hospital group of alterations from a definite diagnosis to "unclassified."

There were some alterations from one organic group to another, as 28451 (general paresis to tabes and tuberculosis), 28456 (senile to organic dementia), 28395 and 28491 (organic to senile dementia). Two were from dementia precox to imbecility (28150, 28227).

The small group sent to the Danvers Hospital shows a superficial discrepancy of opinion in 14 of 29 cases, or 45%. But, as in the case of Westborough diagnosis, a larger part of the discrepancy comes from the "unclassified" group. There were but 22 cases in which the Psychopathic Hospital risked a diagnosis, and there was a change of diagnosis in 5 of these (23%).

From the above analysis it is easy to conclude that the most difficult field in classification is that of dementia precox. It is not, as might be supposed, that the Psychopathic Hospital officers have a bias in one direction. At first we thought that there was a bias. For we found that the Boston Hospital officers had altered our diagnosis of dementia precox in 16 instances, as stated above; and upon review of the cases we saw good ground for the change in at least 11 of these cases. And, when we found that our diagnosis of manic-depressive psychosis had been altered

but once (and then in a case in which the two diagnoses might conceivably be identical), we felt that the Psychopathic Hospital had a true bias against the, as a rule, more optimistic diagnosis of manic-depressive psychosis.

Upon continuing our reviews, however, we found at Westborough that but one case of such an alteration was met with, but that there were 5 instances of alteration of our diagnosis of manic-depressive psychosis to that of dementia precox. At Westborough, too, we found four other cases altered from various diagnoses to dementia precox.

Among the 131 alterations of diagnosis we find the largest single group of alterations to concern the diagnosis of dementia precox or manic-depressive psychosis (42 cases, or 32%). Thirty-five of these cases show a shift of diagnosis from one to the other of these two diagnoses, viz., 20 from dementia precox to manic-depressive and 15 in the reverse direction. Boston yielded four-fifths of the former alteration, and the latter was about evenly divided between Westborough, Taunton and Worcester.

Upon review we consider doubtful or (for external reasons) no longer determinable the issue in 14 cases, leaving 20 which we have more intensively reviewed. In these 20 cases we are inclined to concede the state hospital diagnoses at present.

Eleven of these 20 cases seem to have been excited cases, or cases in which agitation was a feature. We are inclined to believe that the diagnosis of agitated cases is more difficult than that of akinetic cases. It is proposed to throw this problem open in forthcoming diagnostic studies at the Psychopathic Hospital.

Appended are a few condensed histories which struck us as of particular interest.

Three of the five Westborough cases of alteration of Psychopathic Hospital diagnosis of manic-depressive to dementia precox were examples of over-hasty diagnosis at the Psychopathic Hospital. One (10793) should probably not have been diagnosticated at all, as to form of insanity. Another (10634) was partly a language difficulty. A third (10657) illustrates what is so frequently illustrated—the importance of close observation as to hallucinosis.

These three cases may be briefly summarized as follows:—

J. P., Westborough, 10,793 (Psychopathic Hospital 1421) should be placed among unclassified. The case was observed for four days at Psychopathic Hospital, and three days at Westborough before deportation. The diagnosis of the form of insanity must still remain doubtful, although a form of paranoid dementia precox may be suspected. The Psychopathic Hospital diagnosis of manic-depressive insanity was based upon euphoric loquacity, possible flight of ideas, busy interference with all affairs, distractibility. The history secured by Westborough, however, suggests the beginning of a paranoid dementia precox.

A second case might also better have remained unclassified.

S. B., Westborough 10,634 (Psychopathic Hospital 1471), was an illiterate Sicilian, 27 years old, off work six months, said to have been jilted, apathetic, apparently depressed, was thought to show (manic-depressive) retardation during his seven day's observation at Psychopathic Hospital. The *"retardation"* was suspected at the time to be due to difficulties with the interpreter, who did not understand the Sicilian dialect fully. The *apathy* might of course have been that of hebephrenic dementia precox.

In the absence of schizophrenic symptoms, the diagnosis dementia precox has not been fa-

vored at the Psychopathic Hospital. It would have been better probably to have left the case unclassified. At Westborough descriptions were obtained of scenic, dreamlike hallucinations (combined visual and auditory), ideas of reference, delusions involving love affairs and religion, as well as a history of previous spells of excitement. It is still difficult to obtain indubitable evidence of schizophrenia (partly dialect difficulty). To sum up, a Psychopathic Hospital error of diagnosis due to erroneous diagnosis of retardation and lack of ability to get history of hallucinations.

The question of the form of diagnosis must take into account hallucinations of God talking "ever since he (patient) was born," telling him always to "walk straight and harm nobody"; and visions of a church burning up with God on the altar, and the Virgin behind God, and of the Virgin unable to fly to heaven although she wished to; or again of a church, with priest saying mass, and his sister playing the organ and angering him, saying, "Why don't you buy a new suit?"

Possibly this case belongs in Kraepelin's dementia paranoides gravis (1913). The lesson appears to be that hallucinosis is the key-symptom for study, and that apparent manic-depressive features should not be urged as basis for a definite diagnosis.

J. H., Westborough, 10,657 (Psychopathic Hospital 1085), was a case in which seven days' observation at Psychopathic Hospital was not enough to establish hallucinosis or interpret a certain amount of resistivism, as well as refusal to eat. Resistivism, mutism, negativism, verbigeration, constrained postures, hallucinosis, delusions of being killed or burned, developed at Westborough within three months. At times tube-fed, at other times patient

could be got to eat when told not to eat. After seven months mutism became fixed, and has remained so till present writing.

Two further cases may possibly be examples of two of Kraepelin's newer groups, viz., simple depressive deterioration (11062) and the circular excited form (10988). These cases may be abstracted as follows:—

L. B., Westborough 11,062 (Psychopathic Hospital 2100), gave a history of long, gradual onset (four years from Oct. 29) with worry and crying spells over distasteful work, and with some sex-coloring, perhaps a history somewhat more consistent with the diagnosis dementia precox than with that of manic-depressive insanity. Then came ideas of reference, "must be married tomorrow" to a certain man, "heart down in abdomen," etc. There was an appearance of apathy which the Psychopathic Hospital officers thought might be unreal. At Westborough impulsive acts, persistent covering up with bed-clothes, persistent closure of eyes, silly smiling, and persistence of delusion about heart with exhibition of further sexual-religious delusions (married by Jesus to a Portuguese by proxy; identification of female patients as boys whom she had borne in some unusual way).

This case has much in common with Kraepelin's new simple depressive (or stuporous) deterioration, a form of dementia precox. The case has not been stuporous and has the insidious onset of the non-stuporous form of simple depressive deterioration, as described by Kraepelin. But characteristic terminal mental impairment has not yet set in (8 months after the acute outbreak). The case shows the prodromal character change of (four) years' duration described by Kraepelin. Hypochondria, auditory hallucinations, delusions of persecution, crying spells, sexual excitement, general conduct disorder, are also characteristic. Negativism

has now developed, but there is not, as yet, any stuporous tendency like what Kraepelin states to occur in about one-third of cases in this subgroup.

M. H., Westborough 10,988 (Psychopathic Hospital 1873) is a case in which the Psychopathic Hospital diagnosis, after seven days' observations, rested on loquacity, playfulness and general appearance, although there was at times an appearance of listening to voices, and there were occasional statements about being called names. Patient wove relevant remarks into talk, but would not make relevant replies to questions. An idea that "dope," looking and tasting like plaster, is falling from ceiling and walls, might have suggested a paranoid form of dementia precox.

After seven and a half months at Westborough, patient was allowed to leave hospital on furlough with husband. Patient exhibited resistivism, negativism (fell limp on being told to hold stiff), apparently unmotivated laughter and smiling, acts of moderate violence (question of motive), spells of excitement speedily quieted by warm, wet packs (often asked to be kept in pack), hallucinosis (two months after transfer) apparently persistent for four months.

It is possible that this case belongs in the circular excited group of Kraepelin's edition, 1913. The onset was with delusions of persecution, probably on a basis of auditory hallucinosis. Patient was first medically observed in the excited phase which warrants her inclusion in the new group. Distractibility and assimilation of adjacent talk and happenings form a feature suggestive of manic-depressive mania; but these are combined with negativism, impulsive acts, and aimless shifting of attention so as to suggest dementia precox.

Kraepelin states that more than one-half of these cases (53%) have remissions, but these as

a rule appear after the initial depression, not as in this case, after a fairly long excited phase. The total course (exclusive of remissions) is rarely as long as two years. The question of diagnosis will probably be resolved shortly.

At this point it is well to consider the single case in which a Psychopathic Hospital diagnosis of dementia precox was altered by Westborough to manic-depressive insanity.

J. B., Westborough 10,974 (Psychopathic Hospital 1858). This diagnosis of dementia precox was erroneous because the remnants of alcoholic hallucinosis (or of delirium tremens, it is not certain which), superimposed on what was probably a manic-depressive excitement gave the impression of dementia precox, especially as conversation, at first relevant, would tail off into irrelevance and incoherence, which taken by itself gave at times a little of the impression of schizophrenia. A history of chancre complicated the picture. In the second month at Westborough patient had an excited attack or exacerbation of excitement *without* hallucinosis and in four more months was about ready to go home recovered. The error might not have been made, had the extent of the patient's previous alcoholism been known (he afterward told of a still previous touch of delirium tremens), and had the picking of imaginary particles from his arms and hands, shortly after entrance, been given due attention.

Six Worcester cases touch dementia precox problems. Three deserve especial study as being alterations from manic-depressive insanity to dementia precox, thus comparable to five instances in the Westborough Hospital group.

L. J., Worcester Hospital 28,191 (Psychopathic Hospital 386), was at Psychopathic Hospital (1) Oct. 19-Nov. 27; (2) Dec. 3-Feb. 6 (becoming a voluntary patient Dec. 7; (3) Feb. 16-17 (Boston police case) receiving both times the diagnosis of

maniacal manic-depressive insanity. A period of mutism at first, and in the last residence grimacing incontinence during the prodromal period, persistent and assumption of grotesque attitudes, might have given rise to the suspicion of dementia precox. The serum was positive to the Wassermann test at first, later negative; there seems to have been no clinical evidence of syphilis. Patient, while at Worcester, once gave an account of what were possibly auditory hallucinations of a religious coloring, which, she said, came through a ventilator at the Psychopathic Hospital. Patient was committed to Worcester Feb. 17. In March patient had periods of confusion in the midst of continuous excitement, and had numerous superficial abscesses. Comparative quietude and insight until July, when excited. Another excited spell in August (untidy). Apparent blunting (facial expression) and lack of insight. Scattered thinking. Discharged on trial visit Sept. 24, 1913, "improved." March 27, 1914, mother regarded her as well.

A. B., Worcester Hospital 28,963, has mother who has had several depressions, twice in Westborough Hospital. Patient had four attacks of excitement of about three months each between 18 and 35; as well as numerous depressions, not well remembered, and a tendency to hyperreligiosity. At 43 came the fifth attack of excitement (Psychopathic Hospital), in which the most suggestive symptom which might be regarded as one of dementia precox was drinking sputum from another patient's cup. At onset he cut up his clothes with a razor, walked about naked at a friend's house, and did other similarly odd things. Upon commitment to Worcester, stated that he had no passage in his bowels, later that he was a dead man, and expressed various delusions. The Worcester staff thought at first that the case was manic-depressive (possibly dementia precox) but in the course of five months altered the diagnosis to dementia precox. There seems to be no evidence of hallucinosis at any time, or of characteristic catatonia. The delusions remind one of involution-melancholia.

SUMMARY.

The writers discuss the difference between insanity and mental disease. Studies of similar scope at Danvers (general paresis, senile dementia, psychoses in general) and at Worcester (general paresis) are mentioned, and a table is offered showing the high accuracy which the diagnosis of general paresis had obtained even before the Wassermann reaction was available. They remark upon the frequency of "unclassified" cases at Danvers Hospital and show a similar frequency at the Psychopathic Hospital, Boston.

It is shown that about one in five cases get no diagnosis at the Psychopathic, and that, of those cases that do achieve a diagnosis, one in four has its diagnosis altered upon removal to a state hospital. Not all of those removed receive a definite diagnosis. There is, in fact, a residuum of about 6% that have as yet remained unclassified.

Some analysis is made of the figures for five hospitals receiving the majority of the Psychopathic Hospital patients. Possible bias in diagnosis is considered, but largely discarded. The most difficult field of diagnosis is shown to be that of dementia precox and manic-depressive psychosis. It is thought that the excited or agitated patients of these groups form the largest and best subject of diagnostic and theoretical investigation. Examples of interesting alterations of diagnosis are offered, including a case in which the terminal phase of an alcoholic hallucinosis together with incoherence (perhaps due to attention-disorder) and maniacal symptoms was the basis of a diagnosis of dementia precox, whereas the true diagnosis was very probably manic-depressive psychosis combined with alcoholic psychosis.

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XLIII

ADVANTAGES OF A PATHOLOGICAL CLASSIFICATION OF NERVE CELLS

WITH REMARKS ON TISSUE DECOMPLICATION AS SHOWN IN THE CEREBRAL AND CEREBELLAR CORTEX*

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Last year I brought before the Association a communication on the direction of research as to the analysis of cortical stigmata and focal lesions in certain psychoses.[1] Therein I essayed to present some reasons why the present neuropathological situation is not entirely hopeless, even when we approach the fundamental genetic problems of mental disease. I felt that there was much warrant for declaring that the stock of structural investigation, so far from having dropped below par, was indeed a very promising stock. My chief point was that, in the analysis of the cerebral cortex in mental disease, advantage must be taken of the transition planes between "sensory" and "psychic" tissues as provisionally defined by the modern cortex topographers,[2,3] for the purpose of observing the differential reactions of the so-called "sensory" and "psychic" tissues to identical conditions of a disturbing or noxious nature. For these adjacent zones of "sensory" and "psychic" tissue, whatever their embryogenesis or special physical and chemical natures, are as a rule under identical mechanical conditions (*e. g.*, of vascular

*Being contributions of the Massachusetts State Board of Insanity No. 121 (1915, 24).

Bibliographical Note: The previous contribution of the Massachusetts State Board of Insanity's series of scientific contributions was No. 120 (1915, 23) by E. E. Southard, entitled The Feebleminded as Subjects of Research in Efficiency, published in the Transactions of the 1915 meeting of the National Conference of Charities and Corrections held at Baltimore.

supply, of elasticity and pressure, of heat and the like), and may be safely conducted through the enormous gamut of physical and chemical change involved in modern microscopic technic under conditions so nearly uniform that without difficulty the "visuosensory" and "visuopsychic" tissues types, the "auditosensory" and "auditopsychic" tissue types, as well as many other contrasting types, can be observed under the selfsame coverglass. Of course the technical difficulties are greater in the way of comparing two coördinate portions of a given cortex type in the two hemispheres, since the technic of "total" brain sections, while suitable enough for fiber studies of an anatomical nature, is unhappily as yet not suited to the finest studies. However, much can be learned from a systematic comparison of tissue types in the two hemispheres as well as from a study of immediately adjacent cortex types. Perhaps I do not need to insist that the final truth or preciseness of the designations of cortex tissue as "sensory" and "psychic" is not involved in any question as to the value of studying these tissues, since it is safe to say that everyone is now agreed that at least some part of the mind is related with some part of the cortex and that all parts of the cortex are more or less directly related with one another.

Last year I presented a few examples to illustrate my general contention that so-called "sensory" and so-called "psychic" tissues may show a differential pathological reaction despite their adjacency and apparent subjection to identical physical and chemical conditions. My examples were, it may be recalled, from the comparatively well charted visual area (calcarine—"visuosensory," common occipital—"visuopsychic") and the somewhat less well understood common sensory area (postcentral gyrus, susceptible to a similar subdivision).

The conclusion is inevitable, whether or not my special contentions are in detail correct (notably my correlations between "visuopsychic" tissue gliosis and visual hallucinations and between gliosis of an area related to complex kinesthesia and the symptom known as catatonia,[4 5]) that the general contention must remain standing, to the effect that neuropathology has here a method which, however tedious and expensive, is surely rewarding to the

extent of showing something pathologically different in two closely adjacent areas under identical general vascular conditions.

This year I wish to animadvert in a still more general way upon the topic of *microlocalization* in the cortex. Whereas my argument may seem immediately foreign to the internistic aims of the members of this Association, yet I am convinced, (1) that the claims made are claims of a distinctively pathological (and not generally biological) method of study, and (2) that internists are those who do see, or should see, the value and applicability of such essentially pathological (medical) methods to such topics as modern psychiatry.

I know that I should by rights dwell a little on that portion of my title which speaks of the advantages of a "pathological classification" of nerve-cells. We pathologists (taking all physicians for the moment to be pathologists, as in a sense they are well-known to be) are nowadays too prone to regard our methods and classifications as biological and as without further specificity. One might as well, in the opinion of the writer, dispose of physiological methods in terms of physics and chemistry, or the method of physics as a compound of geometrical and phoronomic methods. However that may be, it seems clear that the pathologist has much interest, not only in those metric and statistical methods which modern biology employs, but also in what may be termed "survival values." The term "survival values" has a Darwinian ring and perhaps justifiably so. Yet when we are considering the survival value not of an organism but of parts of organisms, the simple Darwinian aspect of the conception alters. For we have in mind, not so much synthetic effects, as analytic effects. Gowers' conception of differential "abiotrophy" has something in common with the doctrine of distinct differential survival values. To be sure, the Gowers concept has rather more to do with certain tissues that as it were run out or run down than with tissues interfered with differentially by disease. The concept of abiotrophy is rather more a concept of primary or idiopathic ceasing of a tissue to thrive than a concept of a thriving actively stopped or blocked. In brief, the concept of survival values here discussed is rather more a matter of the internal economy of the multicellular metazoan than a matter of Darwinian survival of whole organisms. And, again, the concept

of survival is of survival, not merely in stable conditions (like the running of a clock until it stops) but in unstable conditions (like that of a clock stopped by some external force).

This is not the occasion to discuss the logic of methods in pathology or in neuropathology. It is sufficient to contend that tissues and cells apper to have differential vitalities and that these differential vitalities appear under the guise of a difference, not merely in what Bergson might call the *élan vital*, but also in the powers of resistance of the tissues or cells. Now the latter have not been sufficiently studied in the concrete, especially in neuropathology, despite the fact that neuropathology is supremely well characterized for such study on account of the variety of cells packed away in small geometrical units in the central nervous system. We should be able to study effectively the indices of resistance of nerve cells. A truly "pathological" classification of nerve cells might then be a classification of nerve cells according to their indices of resistance and survival under a variety of conditions. Naturally we are a number of years away from even the more obvious of such classifications. I wish to make it my present task to present a striking contrast in apparent survival value of various apparently homologous elements of the cerebral and the cerebellar cortex.

My conclusions naturally touch, in the present phase of neuropathology, only a few of the cellular elements of the cerebral gyrus and the cerebellar lamina respectively. Moreover, my conclusions as to the curious distinction in power of resistance between the large Betz cells of the precentral gyrus of the cerebrum and the large Purkinje cells of the cerebellar lamina is not founded on a thoroughgoing systematic study of the reactions of these cells under all known physical and biological conditions. A systematic study of a still more general nature is of course highly desirable, although it cannot be carried out in any existing laboratory with the funds at the disposal of neuropathology, a science in general almost inconceivably neglected. Perhaps I would best term my results "impressions" rather than "conclusions," based as they are upon observations confined to such effects of local hemorrhage, intoxication, exudation, and the like, as the natural experiments of human neuropathology permit.

Under ideal conditions, we should doubtless approach the matter of neurones from the geometrical side and describe and measure, name, and classify all available nerve cells in the major types of animals, including man. Such a task has been reasonably well carried out for the more easily available cells of the circulating blood. The necessity of executing a similar plan for fixed tissues has not appealed to the histologists to any convincing extent, and perhaps rather less so in neurology on account of the world's habit of postponing neurological work until other apparently simpler affairs are well in hand. Only of late has a classification of neurones from the standpoint of mechanics been attempted, and even such work as that of Dolley and Crile has suffered from the lack of fundamental metric work done by histologists. It is naturally difficult to evaluate the various metamorphoses of the Purkinje cells which Dolley and Crile are inclined to regard as of dynamic significance when so little is known as to the mechanical and physicochemical conditions under which nerve cells of all sorts must labor. Accordingly, it seems unsafe to draw extensive conclusions of a physiological or neuropathological nature from what we at present know of neurocytology, whatever opinion we may hold as to the objectivity of the Purkinje cell findings of recent American workers. There can be no doubt, however, that the Purkinje cell is a peculiarly feeble cell under a number of conditions which can be in part defined. There can be no doubt that Dolley, Crile, and their followers, have selected a cell of considerable lability; that is, a cell with a comparatively low index of resistance to at least certain disturbing or noxious influences.

I had occasion a number of years ago to observe the extraordinary lability of the Purkinje cells in some work on the neuroglia framework of the cerebellum in cases of marginal sclerosis[6] (1905), and again in some work on lesions of the granule layer of the human cerebellum communicated at the meeting of the American Association of Pathologists and Bacteriologists in 1906.[7] At that time I noted that Obersteiner,[8] so long ago as 1871, and Kirchhoff[9] in 1882, had described absence of Purkinje cells from atrophic areas in the cerebellum. In fact, in my second paper on the cerebellum above mentioned, I noted as a "familiar fact" that the great

elements of the Purkinje cell belt are the first to undergo destruction. Interest in those days was directed rather at neuroglia reactions than to essential changes in the Purkinje cells themselves. In that older discussion, I pointed out that in many types of lesions the Purkinje cell belt is the initial seat of activity, which activity takes the form of neuroglia cell overgrowth. The Purkinje cell layer must be in fact regarded as a *locus minoris resistentiæ*. In the present year I was pleased to observe in the course of some work[10] done in the Psychopathic Hospital Laboratory by Dr. Egbert W. Fell, the striking tendency to the deposit of fat (demonstrable by the Scharlach R method) in and about the Purkinje cells in cases of mental disease apparently due to intoxication or infection (the group of so-called "toxic" psychoses), although Dr. Fell was not able to discover similar intracellular changes in a variety of other more chronic conditions. There is good reason then why modern workers should choose the Purkinje cells of the cerebellum as a favorite field for the study of the effects of a variety of changed conditions.

The point of my present communication is that the large Betz cells of the precentral gyrus, taken as representing the motor cortex, are far less labile than the Purkinje cells. The likelihood that Betz cells would withstand relatively tremendous physico-chemical changes was more or less consciously described in my paper of 1908 on the "Mechanism of Gliosis in Acquired Epilepsy."[11]

In casting up a program for the better development of neuro-pathological research, I could conceive no better program than the investigation of neurones from the standpoint of their differential lability. Expressed otherwise: What is the index of resistance? What is the vital capacity? What the viability or "survival value" of the different cells? In default of a complete geometric classification, in the absence of any signs of a phoronomic classification (indeed, such phoronomic classification is unlikely to have great scope in the cells of fixed tissues), in the prevalent feeling of grave doubt as to the virtues of a mechanical classification (whether this be taken from the standpoint of equilibrium or from a kinetic standpoint), we must turn perhaps to a more concrete method of classification. There are some signs of feasibility perhaps in the

tinctorial classification proposed by Nissl, although this eminent worker is far from considering his tinctorial classification as based on anything but what he terms "cell equivalents." Aside from this endeavor of Nissl, which logically derives from Ehrlich's proposal of methylene blue as an agent by which specific characters of cells might be observed, there are few or no traces of physical and chemical classifications in any stricter sense. Thus, should one inquire whether the elasticities of nerve cells have been studied or whether we know much concerning the effects of pressure upon nerve cells or the phenomena of their so-called "capillary" chemistry, we should find an almost Egyptian darkness to prevail. Something, to be sure, is known concerning the thermal metamorphism of cells, at least the disruptive and destructive effects of heat. The various other branches of physics have been very largely neglected by the neurocytologists.

To illustrate the difficulty of the situation, I have been accustomed to quote from Sherrington a laborious catalogue of changes which might occur at the synapses, which Sherrington himself defined.[12] In the light of this catalogue, which I give below, the condition of neurocytology might be regarded as a desperate one; still, if the physical possibilities of the synapses can be defined even to this degree, the outlook is really not desperate. The synapses, according to Sherrington, "might restrain diffusion, bank up osmotic pressure, restrict the movements of ions, accumulate electric changes, support a double electric layer, alter in shape and surface-tension with changes in difference of potential, alter in difference of potential with changes in surface-tension or in shape, or intervene as a membrane between dilute solutions of electrolytes of different concentration or colloidal suspensions with different sign of charge." Accordingly, what chance might a "pathological" classification of neurones possess? At first sight, a negative answer would be given by almost any biologist. Before attacking such a topic from the pathological point of view, should there not be a congress of specialists, if not in geometry and phoronomics, then at any rate in mechanics, physics, and chemistry, which congress should lay out a program of metric and quantitative work upon which conclusions of a pathological nature might properly at some future date be grafted?

In point of fact, the situation of the pathologists is not so hopeless. The pathologists' work need not be postponed. As I have elsewhere[13] and in a quite other connection been emboldened to state, pathology has its short cuts. It is perhaps a science of short cuts to biological conclusions. For example, it would be difficult to estimate how many decades it would have taken a congress of workers in physiology and in normal cytology, to discover the Virchowian principle, *omnis cellula e cellula*. From a study of exudation, Virchow was led to his interesting generalization after a comparatively brief period of work. Another instance may suffice from an entirely remote field. A tremendous impetus to the study of human character has beyond question been given by William James's *Varieties of Religious Experience*, a book which may be said deliberately to adopt the study of caricatures of character as the best means of coming at a reasonable notion of normal character types; at least, such I believe to be the trend of psychobiological study since the publication of that work. To be sure, the task of describing and evaluating character is (if possible) still farther from completion than the task of cellular biology in general and of neurobiology in particular.

The logic of pathology has nowhere been comprehensively studied so far as I am aware; even so gigantic a work as that of Wundt on *Logik*, written by a man of great physiological as well as psychological insight, fails to consider systematically this topic. It may well be, however, that the specific logic of pathology, as far as it is not identical with that of physiology, physics, and chemistry, is somewhat dependent upon the theory of survival values of cell units. The death point of a cell under varying conditions is a far more definite datum for study and interpretation than the majority of quantitative variations which the students of the normal histology, physiology, and the like are able to contribute.

However true or relevant all this may be, there surely does attach a certain intrinsic interest to studies in the death point of nerve cells. Take, for example, such a question as the nature of those almost entirely obscure mental diseases known as dementia precox and manic depressive psychosis, in the latter of which

diseases singularly little can be observed in the gross brain.[14] Let us suppose the indications of a special line of attack upon the histology of manic depressive psychosis (as presented in my communication[15] with Bond in 1914) to be sound. Still the pigmentation or lipoid deposits of the cells would remain merely quantitatively different from those found possibly under normal conditions or in conditions of senescence. Far more convincing here would be such results as those of Orton in his study of satellite cells in fifty selected cases of mental disease.[16] For the occurrence of satellite cells (so-called satellitosis) in the nervous system in increased number doubtless argues far more than the stationary changes that are normal to nerve cells, far more than mere alterations in the "self-supply" of their metabolism. In fact, perhaps satellitosis or neuronophagia may be regarded as definitely denoting a certain disappearance of elements or portions of elements normal to nervous tissue. At least such might be our best hypothesis in the light of the contentions of Weigert notably in his studies of the neuroglia[17] in 1895.

The contentions of my communication before the Association last year (as to satellitosis, or overgrowth of a certain kind of neuroglia cells in the so-called "visuopsychic" area when no such overgrowth was demonstrable in the immediately adjacent "visuosensory" area) are obviously based upon identical conceptions. To throw into sharp focus the importance of such a comparative study of death points of cell types in the cerebrum and cerebellum respectively, I will reproduce two diagrams of the neurone supply of the cortex cerebri and of the cortex cerebelli respectively, as used for some years in my undergraduate course in neuropathology. A diagram of a similar nature, involving other neurones, has been before published in a paper on the optic thalamus.[18] The principle of these diagrams was communicated by Dr. S. T. Orton and myself at the meeting of the American Association of Pathologists and Bacteriologists in 1913, but has remained unpublished.

Fig. 1 deals with the neurone supply to the cortex cerebri, and is largely based upon prevalent ideas as to the course of impulses in the cerebral cortex, taking the precentral gyrus as the best known tissue. The diagram enables one to dispense with the

schematic semi-diagrammatic pictures found in most text-books and based upon more or less thorough idealization of actual pathological conditions. The ideas expressed by the diagram follow rather more closely the ideas of von Monakow in his *Gehirnpathologie* of 1909 than those of other authors. The arrows indicate the possibility of numerous short cuts which may well correspond with short

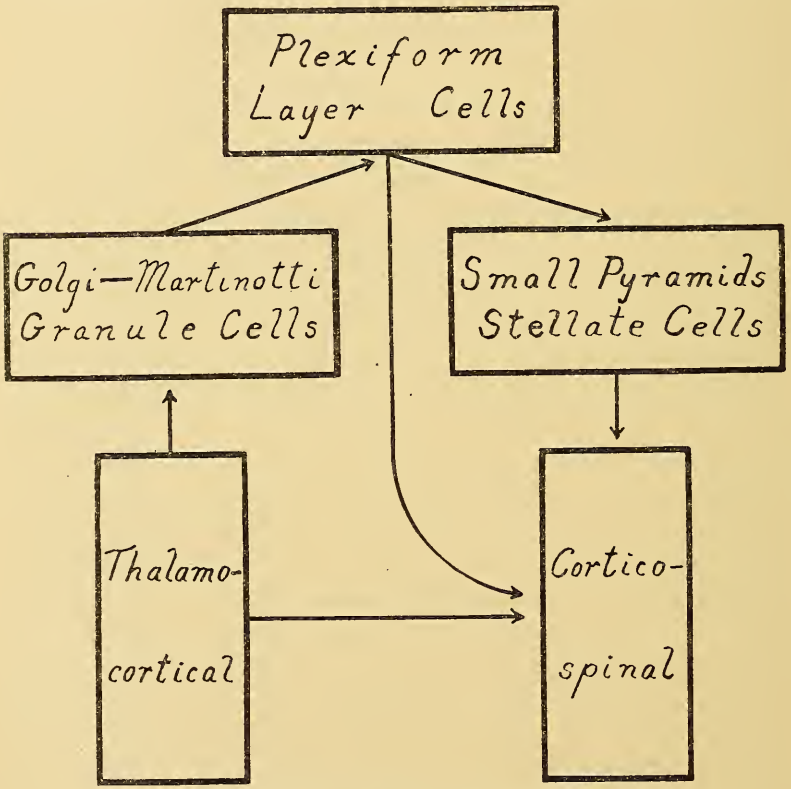

FIG. 1.—Neurone supply of cortex cerebri.

cuts actually used in function. The oblong unit containing the term "corticospinal" is of course the neurone whose cell body has long been known as the giant pyramid or Betz cell. If, now, an impulse passes through the "thalamocortical" unit so-called (the question of whether impulses run directly or by two or more units from the lower centres to the precentral gyrus is not essential

to the point at issue), then it may well be that sundry dendritic connections permit impulses to be passed directly into the Betz cell, as indicated by the arrow running from left to right from the unit named "thalamocortical" to the unit named "corticospinal." Possibly such a course of impulse is normal for certain relatively fixed and habitual neural processes. However, other paths are possible and, the higher the evolution of the animal, the more elements exist through which complicated paths may cut themselves. The majority of authors now consider that the granule cell layer or stellate cell layer divides the tissues of older evolution below from the tissues of more recent evolution above. It is regarded as an approximation of truth to say that the entire supragranular (suprastellate) system of paths may be regarded as related with higher functions. At any rate, various lower animals do not possess any truly supragranular tissue whatever, and possibly this fact is as good an index of their "inferiority" as any other.

Now it is clear that the corticospinal neurone (giant pyramid, or Betz-cell neurone) is in some ways the most important neurone in the precentral (motor) gyrus, since it is, as it were, the discharging neurone of said gyrus. If any impulses whatever can arrive at the corticospinal neurones, then some sort of action must result provided that the lower mechanisms are intact. Some sort of action or defensive manoeuver, whether of extension or flexion, whether a blow or a tremor, locomotion or static posture, must result. Let, however, the Betz cells be destroyed and the impulses that arrive or are initiated within the gyrus will fail of their effect, or will secure an effect through very devious paths. How important, then, may it well be to the economy of the organism that of all the cells of the gyrus, the giant cells shall possess the highest index of resistance! This the Betz cells seem to possess, at least so far as the fragmentary data now available can prove the point.

Let us now contrast conditions in the cerebral cortex with the somewhat simpler conditions of the cerebellar cortex. Fig. 2 deals with the much simplified neurone supply of the cerebellar cortex following some of the ideas of Ramón y Cajal. If we suppose that impulses arrive in the cerebellar lamina by way of the climbing fibers or moss fibers of Cajal, it is an easy speculation to conceive

that far more fixed and habitual reactions may be secured by way of the direct effects of the climbing fibers rather than by the mediated effects of the moss fibers. For the moss fiber neurones must by hypothesis send their impulse to the Purkinje cells by way of the granule cells.

On the score of function, it would seem that the giant cell of the cerebral cortex and the Purkinje cell of the cerebellar cortex must be homologous cells and together regarded as the discharging cells of their respective neurone complexes. It would be natural to suppose, then, that nature's neural economy might best be conserved by the maintenance of the Purkinje cells at a high index of resistance. Why should not the Betz cells and the Purkinje cells possess approximately identical indices of resistance?

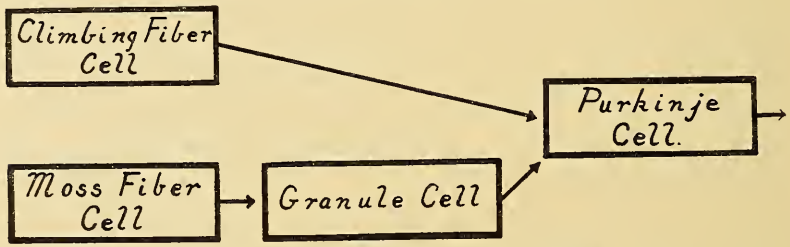

Fig. 2.—Neurone supply of cortex cerebelli.

Much research needs to be done along the indicated line, and it is clear that the earliest researches to be completed along these lines would very possibly deal with other elements roughly approximating in size the Betz cells and Purkinje cells. I do not wish to maintain that under all conditions which might be imagined the Betz cell is more resistant than the Purkinje cell. There may very well be differential physicochemical conditions in which the Betz cell would prove to be less resistant than the Purkinje cell. Nor is it my purpose here to explain in detail the evidence for the particular contention concerning the respective viabilities of these two cell types. The point has been sufficiently emphasized that there may exist such differential viabilities of important nerve cell types.

A word more as to the functional interest of the demonstration

of such viabilities. There seems to be little doubt that the supragranular (suprastellate) tissues are the point of attack in many cases of epilepsy. As I tried to express the matter in 1908, "The outer nerve layers tend, so to say, to *weather out* more readily than do the inner layers when under virtually the same conditions with respect to noxious agent." A number of cases in the study of gliosis in acquired epilepsy seem to support the conception that tissue destruction to a moderate degree may frequently leave the discharging apparatus intact when the inhibitory and controlling apparatus is destroyed.

If we should simplify (or as I now prefer to say "decomplicate") the tissue, we bring about the conditions for unimpeded discharge (a phrase of Hughlings Jackson). In the progress of convolutional atrophy in certain cases of epilepsy, the best conditions for lateral discharge from cell to cell seem to be produced so that we may well imagine that we may fail to demonstrate the existence of sufficient dendritic connections between the intact giant cells so that favorable conditions are produced for the so-called "march of convulsions."

Seeing that the small cells of the cortex have been removed by a process of "decomplication" and the phylogenetically older cells, the Betz cells, remain working, the precise condition is afforded for a sort of automatization of the cortex. In later work in connection with a study of the optic thalamus, I spoke of the simplification of the cortical tissues found in various cases of organic epilepsy as favorable to the march of convulsions, and spoke of the tissues as reduced to conditions not much more complex than those which prevail, say, in the spinal cord. "Impulses once initiated in the neurones of such a spinalized cortex ran off with the speed and inevitability of spinal impulses when upper inhibitors are cut off, and a kind of hyperkinesis, namely, the epileptic attack, ensues." To be sure, a great number of adjuvant hypotheses are necessary to the full explanation of epilepsy. Still, if the agents that simplify or "decomplicate" the convolutions in epilepsy were to simplify by means of destroying the Betz cells, or even by destroying all possibility of functional communication between Betz cells, then epilepsy might not exist. If the sort of thing were going on in the cerebral cortex which seems to go on under identical

conditions in the cerebellar cortex, then, by hypothesis, under these conditions epilepsy might not exist.

Let us turn to the cerebellar situation. Here, as we have stated, it is a familiar fact that the Purkinje cell layer is a *locus minoris resistentiæ*. As I pointed out in 1905, the macerating cerebellum in edema tends to split along this layer. There is in tuberculous meningitis and in epidemic cerebrospinal meningitis often considerable cell débris and edema in the layer. In tuberculous meningitis we not seldom find a focal destruction of Purkinje cells together with a loosening of structures adjacent, and in ischemic injury to the lamina, we may find a line of neuroglia cells in and near the Purkinje belt, as the only sign of reaction for a considerable distance on either side of the layer. A case of typhoid fever with small hemorrhages in the cerebellar pia mater, published in the granule layer study of 1907, offered a natural experiment to prove both the maximum lability of the Purkinje cells and the initial neuroglia activity of the Purkinje cell belt. Pictures consistent with these descriptions have been found from time to time in work since those earlier publications. Evidence seems to prove that in a great number and variety of conditions, particular laminæ of the cerebellum may be entirely closed off from neural function through the death of Purkinje cells despite the persistence of the other smaller cells of the lamina. It would seem that of two lesions which seem to decomplicate the cerebral and cerebellar cortex equally, the one leaves the Betz cells intact and the other destroys the Purkinje cells. The decomplication in the one case reduces the cerebral cortex to conditions of an automatic machine by a process we have named "spinalization;" in the other case, cerebellar function is "bottled up" through the destruction of the discharging elements of the lamina.

Of what advantage this distinction might be to the organism, it would be premature to say much. Nevertheless, it might be said that it would be of some general survival advantage to the organisms to preserve its Betz cells as long as possible, even if the remainder of the cortical apparatus were reduced to archaic conditions. In the case of the cerebellum, on the other hand, it might well be asked whether the function of the total neural mechanism might

not be still more unfavorably affected if the Purkinje cells were to persist when the granule cells were dead. The cerebellum in that event might seriously perturb the action of the neural mechanism by throwing unfortunate tonic, sthenic, and metric factors into a given functional situation. It seems to be generally thought that the cerebrum can get on fairly well without the cerebellum, and that in the higher organisms at least the cerebellum is of lesser value taken by itself. If the cerebrum has more of a function with respect to the sequence and succession of muscular events, then the cerebellum has more to do with the pattern and simultaneous correlation of muscular activity (tone, intensiveness, coördination, and the like). But if we may go farther and say that the cerebrum is itself also capable of undertaking with fair success the duties of coördination in space, then it is clear that the cerebellum is more of an adjuvant of the cerebrum than the cerebrum to the cerebellum. If this account is approximately true, then it might be of evolutionary value that the Purkinje cell should be blotted out under precisely the same conditions in which the Betz cell should be maintained.

We may add another point if we like to the effect that, so far as we know now, the cerebellar cortex is almost entirely uniform in its general structure throughout, and that at present no means exist of telling one lamina from another. If this is the case, then a gradual cerebellar destruction involving the Purkinje cells of a lamina or two might not interfere with the proper workings of the tonic and sthenic functions of the cerebellar cortex; for if only the Purkinje cells of a lamina are placed *hors de combat*, then by lateral conduction through numerous intact cell connections, impulses might reach intact Purkinje cells and thereby reach the dentate nucleus to be distributed safely enough to proper destinations. Perhaps, however, this latter speculation would better be entirely suppressed at the present time.

Enough has been said to show that neuropathological research:

1. Might do well to engage on a program of studying by available methods the differential viabilities of the various nerve cell types, thereby erecting an essentially "pathological" classification of nerve cells on the basis of their powers of resistance or survival values; and

2. Might endeavor to collect data as to the differential effects of simplification or "decomplication" of nerve tissues, having in mind the evolutionary or survival values of the functions which different forms of decomplication would destroy or leave intact.

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XLIV

THE SIGNIFICANCE OF BACTERIA CULTIVATED FROM THE HUMAN CADAVER: A SECOND SERIES OF ONE HUNDRED CASES OF MENTAL DISEASE, WITH BLOOD AND CEREBROSPINAL FLUID CULTURES AND CLINICAL AND HISTOLOGICAL CORRELATIONS.*

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ABSTRACT.

Problems of autopsy bacteriology in the insane.
Conclusions of Gay and Southard (1910) concerning bacteriology of blood and cerebrospinal fluid and concerning lipoid changes in the nervous system.
Special features of the Boston as compared with the Danvers material.
Summary of technic employed.
Percentages of sterile cultures (various authors).
Cultivation with respect to number of hours post-mortem (Tables I. and II.).
Species of bacteria found (Tables III. and IV.).
Species of bacteria found in bodies of general paretics (Tables V. and VI.).
Cases with blood and cerebrospinal fluid sterile (Tables VII. and VIII.).
Bacteriology of cases without diffuse lipoid changes (Tables IX. and X.).

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* Being contributions from the State Board of Insanity, Massachusetts, Number 24 (1914.4). The work has been aided in part by the Underhill Gift to the Department of Neuropathology, Harvard Medical School, for the study of non-nervous factors in mental and nervous disease. (*Bibliographical Note.*—The previous S.B.I. Contribution, Number 23 (1914.3), was entitled "On the nature and importance of kidney lesions in psychopathic subjects: A study of one hundred cases autopsied at the Boston State Hospital," by E. E. Southard and M. M. Canavan, published in the Journal of Medical Research, Vol. XXXI., No. 2, November, 1914). The bibliographical note to the contributions just mentioned was in error as to the previous S.B.I. Contribution on page 285 of Volume XXXI., No. 2, November 1914, which should have read as follows: "The previous S.B.I. Contribution (Number 22, 1914.2), by E. E. Southard, was entitled 'Notes on public institutional work in mental prophylaxis, with particular reference to the voluntary and temporary care admissions and the "not insane" discharges at the Psychopathic Hospital, Boston, Mass., 1912–13.'" Accordingly, the contribution on pages 285–299 of the volume just mentioned should have read (Number 23, 1914.3). Received for publication July 27, 1914.

The bacteriology in cases with terminal disease having assigned duration less than four days (Tables XI. and XII.).

Analysis of nine cases each showing the same organism in blood and cerebrospinal fluid (Table XIII.).

Lipoid changes in these nine cases.

Conclusions: (a) compared with those of Gay and Southard (1910), (b) epicritical.

A study[1] quite similar in scope to this was published by Gay and Southard in 1910 from material in their services as bacteriologist and pathologist respectively at the Danvers State Hospital, Hathorne, Massachusetts. The actual technical work of the 1910 study was carried out, so far as bacteriology was concerned, in the years 1906–1907, and was shared in by Drs E. T. F. Richards, Anna H. Peabody, and Myrtelle M. Canavan. During that and subsequent periods several special bacteriological studies were made upon Danvers material, both for systematic investigation of routes of invasion by the bacteria found (mesenteric lymph nodes,[2] bronchial and retroperitoneal lymph nodes,[3] pelvic lymph nodes[4]) and for more specialized purposes.[5,6] One brief case-study was also published from the Boston State Hospital.[7] All this work, together with findings incidental in the Danvers investigation of bacillary dysentery[8,9] and a study (antedating these) by Hoag on certain diphtheroid organisms,[10] may be thought to give a fair picture of bacterial findings by prevailing aërobic methods in the bodies of the insane after death, as far as Danvers hospital was concerned.

There was always the suspicion, however, that the Danvers conditions were somehow atypical. Would another laboratory with similar technic yield similar results? Climatic, dietetic, epidemic, sanitary, and various environmental differences, to say nothing of differing classes and ages of patients, might in some other institution well yield different results as to bacteria found in cadavers. The percentages might be lower or higher, or they might remain approximately identical. The species of bacteria found might be quite different in a new locus with a fresh prevailing flora in the environment of wards or laboratory.

When one of the writers (M. M C.) took charge of the laboratory of the Boston State Hospital it was determined to institute a similar investigation there to the one carried out in Danvers in the years 1906–1907. The laboratory of the Boston State Hospital was organized late in 1910, and from the first the physicians there took an active interest in clinical pathology, in autopsy correlations, and especially in hygiene. The investigation of various small epidemics gave a bacteriological atmosphere to the laboratory, somewhat unlike that of the usual insane hospital laboratory, and indirectly favored this work.

Although the work thus entailed is arduous in the extreme, it has the advantage of a routine sweetened by the prospect of some result (positive or negative), and also of aiding in the solution of numerous individual case-problems.

The problems held in mind during this work were in the main these:

1. Which of the conclusions of Gay and Southard, 1910 (see below), could be substantiated, and which not?
2. The proportion of positive and sterile bloods and cerebrospinal fluids.
3. The relation of positive bloods and fluids to acute degenerative processes in the nervous system (fatty changes).
4. The significance of sterile cases.
5. The relation of cultivations to the number of hours post-mortem which had elapsed when the plantings occurred.
6. The question of ward and laboratory "epidemics" of various non-pathogenic organisms.
7. The question of bacteria having more special relations to certain cases.

To render more concrete the objects of the work, the conclusions of Gay and Southard, 1910, may be recapitulated:

1. The results of bacterial cultivations from the heart's blood and the cerebrospinal fluid post-mortem in 100 cases of mental disease have been correlated with the histopathological findings (Marchi impregnations of spinal cord at three levels) and the clinical histories, having special

reference to a history of terminal disease over or under four days' duration (regarded as a period in which typical Marchi alterations might ensue).

2. The bacteria were cultivated upon agar plates inoculated with 1 to 1.5 cubic centimeters heart's blood and others with the same amount of cerebrospinal fluid. The cerebrospinal fluid was removed from the third ventricle through the infundibulum, severed at its origin.

3. Forty-one per cent of our heart's blood cultures remained sterile (compare Gradwohl, 22 per cent; Otten, 42 per cent; Simmonds, 48 per cent).

4. Twenty-eight per cent of the cerebrospinal fluid cultures remained sterile.

5. Under the conditions of our laboratory the statistics show that there is no significant difference in the percentage of positive cultures from either source at varying hours post-mortem, and that the danger of contamination must be limited to a brief interval after death.

6. Our findings point definitely, if indirectly, to the intravital significance of the bacteria found, despite the fact that in no particular instance is the chain of evidence complete.

7. Since the same bacteriolytic substances are found in the blood serum both before and for some time after death, there is no reason for supposing that bacteria can grow better post-mortem.

8. We now show that bacteriolytic substances are absent in the cerebrospinal fluid, so that there appears to exist therein no extracellular mechanism for the disposal of bacteria.

9. The facts just stated (7 and 8) may account for the higher percentage of organisms in the cerebrospinal fluid.

10. Among the facts concerning the incidence of bacterial forms are these: Cocci were found in the blood in 26 cases; in the cerebrospinal fluid in 34 cases; streptococci in the blood, 8 times; cerebrospinal fluid, twice; pneumococci, blood, 3 times; B. coli aërogenes group, blood, 11 times; cerebrospinal fluid, 25 times; B. proteus group, cerebrospinal fluid, 7 times.

11. The absence of diphtheroid organisms from our series is noteworthy, since in previous years cultivations at the Danvers Hospital had yielded such organisms in several cases.

12. Cultivations from thirteen general paretics are listed; in the positive cases cocci prevail.

13. Nine bacteriologically negative cases are listed; reasons are adduced for certain histopathological changes, possibly independent of bacteria, in these cases.

14. Ten cases which failed to show specified histopathological changes are listed, from which it appears that Bacillus coli is not found associated with such cases unless the terminal disease happens to have been brief. On the other hand, cocci are a frequent finding in this group.

15. Thirty-one cases which had terminal symptoms less than 4 days in

duration are listed; 29 per cent of these failed to show spinal cord degenerations by the Marchi method (as against 10 per cent in the total series).

16. Of the 10 cases selected as showing most numerous spinal fatty degenerations (diffusely scattered blackenings in white and gray matter), 9 showed Bacillus coli communis either in heart's blood or in cerebrospinal fluid, or in both, and 8 in large numbers.

17. Of 18 cases yielding 40 or more colonies of Bacillus coli communis from one or each source, 8 showed extreme degrees of Marchi degeneration, 5 relatively severe changes (intraspinal and intraradicular), and the 5 remaining cases showed considerable intraspinal change.

18. Of 13 cases showing generalized softening of the brain tissue (general encephalomalacia), 10 yielded Bacillus coli communis.

19. A definite relation must be assumed to exist between Bacillus coli communis or its toxine and nerve fiber degeneration.

As a foil to this series from the Danvers State Hospital it is well (as above mentioned) to compare the results of another State Hospital, using the same technic, and the first one hundred results are here presented. The material was unselected and, being drawn from the wards of the Boston State Hospital, was quite comparable with that of the Danvers clinic, inasmuch as the patients were afflicted with chronic mental disease and died of terminal exhaustions or infections. Bodies here have frequently lain at room temperatures from one to eighteen hours (owing to separation of buildings) or at best, at a temperature of 39° Fahrenheit, that of the ice-cooled morgue, while those at Danvers were transferred almost immediately to the morgue at 0 Centigrade (the block architecture facilitates rapid delivery of bodies to the morgue, and the artificial ice plant keeps the temperature approximately at 0 C.).

The age of cases autopsied is on an average higher than that at the Danvers Hospital, being 56 for the one hundred and sixty-one autopsied cases at the Boston State Hospital and 49 for a series of one thousand cases at Danvers. The reason for the high age average is obviously one of sentiment in the community from which this body of patients is drawn. The older the patient, the fewer near relatives who object, and the younger the patient, the less available for post-mortem purposes, because more recover and more have

near relatives who demur. The post-mortem examination was, unless (as in a very few cases) limited permission was given, uniformly complete. It begins with body measurement and description of external appearances, with special stress on nutrition, palpable lymph nodes, decubitus. The trunk organs have been removed, weighed and described, sections cut from each organ, and the brain, pituitary, Gasserian ganglia and middle ears and spinal cord examined and preserved.

TECHNIC FOR ROUTINE POST-MORTEM CULTURES.

Withdraw 1.5 centimeters blood from seared right auricle, and 1.5 centimeters cerebrospinal fluid through seared pituitary stalk; and from any seared and punctured surface that should be investigated. (Usually any parenchyma that is apt to yield results will do so with 3 platinum loop transfers. If dry, use a loop of media to ease the curettage).

Plate from shake tubes of agar agar and incubate for 24 hours.

Transfer to agar slants end of 24–48 hours.

Re-transfer from agar slants to milk, Hiss semisolid, Pepton water, gelatin, bouillon, after 24 hours. Stain organisms by Gram.

Read results from milk, Hiss semisolid, and bouillon on third day.

Read results from indol 3d–10th day.

Read results from gelatin 3d–30th day.

Chester's Determinative Bacteriology was followed in tracing the organisms. The first comparison of results occurs in the percentage of sterile cultures from the heart's blood.

Gradwohl	22% (1904)
Simmonds	48% (1904)
Otten	42% (1906)
Gay and Southard	41% (1910)
Canavan and Southard	44% (1913)

Twenty-one cases showed the cerebrospinal fluid negative. One hundred minus eleven contaminated cases equals eighty-nine, of which 23.6 per cent were negative, leaving sixty-eight out of eighty-nine cases or 76.4 per cent positive. (Gay and Southard, 1910, found positive in cerebrospinal

fluid, seventy-two per cent; Canavan, 76.4 per cent; Tomlinson,* 72 + per cent.)

The tables giving the hours post-mortem and the percentage of patients giving positive cultures are next:

TABLE I. (From Gay and Southard, 1910.)

Percentage of cases giving positive cultures.

Hours P.M.	No. of Cases.	Heart's Blood.	Cerebrospinal Fluid.
0–2 hours.....	11	73%	82%
2–4 "	17	46" } 67%	75" } 80%
4–6 "	6	83" }	83" }
6–12 "	17	67"	58"
12–24 "	30	63"	86"
24–48 "	11	82"	82"
48 + "	8	38"	63"
Total.....	100	Average, 65%	Average, 76%

* H. A. Tomlinson in a preliminary report upon the bacteriological examination of the cortex and the cerebrospinal fluid in forty-seven cases of insanity (Am. Jour. Insanity, October, 1897): Tomlinson reports thirty-four cases of terminal dementia, six senile insanity, four paretics, and three acute delirium.

The technic is not described, nor are hours post-mortem or age of patient stated. Heart's blood was not investigated bacteriologically.

3 cases of acute delirium,	3 + growths.
4 " " general paresis,	3 +
6 " " senile insanity,	3 +
34 " " terminal dementia,	25 +
	34 out of 47, or 72%.

Gay and Southard failed to note this older work in their paper of 1910.

TABLE II. (1913.)

Hours P.M.	Total No. of Cases.	Cases Heart's Blood.	Per Cent.	Cerebrospinal Fluid (Total No. Cases).	Per Cent.
0–2	4	0	0	2	50
2–4	18	12	67	14	77 (57)
4–6	9	4	44	4	44
6–12	25	15	60	15	60
12–24	35	19	54	25	71
24–48	5	3	60	4	80
48+	4	3	75	4	100
Total No. of Cases	100	56	.....	68	

The average per cent of positive cultures from heart's blood was sixty-five in Gay-Southard series and fifty-one in the Canavan-Southard series; seventy-six was the average in the Gay-Southard cerebrospinal fluid series and sixty-eight in the corresponding Canavan-Southard series.

The significant thing about this comparison is the same relative percentage of positives from cadavers preserved under different conditions of temperature. If bacteria are post-mortem invaders, why not more positives from cadavers which were not ice-cooled?

A comparison was made between the results in cases brought immediately to the morgue (within one-half hour) after death and results in cases with delay of six hours or more: In both series about one in every two heart's bloods was sterile (fifty-two per cent in early-to-morgue cases, fifty per cent in late-to-morgue cases). In the case of the cerebrospinal fluids, the early-to-morgue cases yielded also about one sterile to every two; but the late-to-morgue cases yielded only about one sterile to every three cases.

TABLE III. (Gay and Southard, 1910.)

Incidence of certain species of bacteria in 100 autopsied cases.

Organisms.	Heart's Blood.	Cerebrospinal Fluid.
Pyogenic micrococci	26 cases.	34 cases.
B. coli aërogenes group	11 "	25 "
Streptococci	8 "	2 "
Pneumococci	3 "	0 "
B. proteus group	0 "	7 "
B. mucosus capsulatus	2 "	1 "
B. pyocyaneus	0 "	1 "
Unidentified micrococci	8 "	6 "
Unidentified bacilli	13 "	14 "

TABLE IV. (1913.)

Incidence of certain species of bacteria in 100 autopsied cases.

Organisms.	Heart's Blood.	Cerebrospinal Fluid.
Cocci	38 cases.	43 cases.
Bacteria	14 "	14 "
Bacilli	9 "	25 "
Cladothrix	1 "	2 "

Thus cocci prevail in both series.

TABLE V. (Gay and Southard, 1910.)

Bacteriological findings in autopsies of general paretics.

Aut. No.	Sex.	Age.	Hours P.M.	Heart's Blood.	Cerebrospinal Fluid.	Brain Tissue (Broca).
1084 . . .	F.	46	9	Contamination.	Micrococci not identified.	Sterile.
1087 . . .	M.	44	8	Staph. albus.	B. coli communis.	Staph. albus.
1119 . . .	M.	47	21	Sterile.	Staph. albus.	Micrococcus not identified.
1122 . . .	M.	35	23	"	" "	Sterile.
1123 . . .	F.	28	6	"	" "	"
1126 . . .	M.	47	4	"	Micrococci not identified.	"
1129 . . .	F.	37	36	Staph. albus.	Sterile.	"
1131 . . .	M.	35	6	Bact. tenue.	M. concentricus.	"
1141 . . .	M.	45	2	Staph. albus.	Staph. albus.	"
1147 . . .	M.	45	1	Pneumococcus.	Staph. cereus.	"
1157 . . .	M.	44	6	Streptococcus.	Sterile.	"
1161 . . .	M.	42	16	Sterile.	B. coli communis.	"
1180 . . .	M.	40	17	Staph. cereus.	Sterile.	"

TABLE VI. (1913.)

General paralysis and cerebral syphilis.

Number.	Sex.	Age.	Hours P.M.	Heart.	Cerebrospinal Fluid.
1911- S . .	F.	48	15	Sarc. pulmonum.	M. pyogenes.
1911-11 . .	F.	35	3	Strep. sanguinis.	O
1911-13 . .	M.	43	13	O	O
1911-14 . .	F.	43	6	O	M. lactis.
1911-16 . .	M.	57	5	Bact. influenzæ.	B. Wesenbergii.
1912- 9 . .	M.	36	3½	M. cereus.	M. carneus.
1912-13 . .	M.	54	16	O	O
1912-15 . .	M.	59	12	O	Sarc. aurantiaca.
1912-20 . .	M.	54	18	O	Contamination.
1912-43 .	M.	52	16½	Bact. aërogenes.	B. vulgatus.
1912-46 . .	M.	59	14	B. vulgatus.	—
1912-49 . .	F.	52	8	M. pyogenes.	—
1912-53 . .	M.	52	19	Bact. truncatum.	M. expositionis.
1912-54 . .	M.	46	3	O	Bact. limbatum.
1912-58 . .	M.	53	16	O	M. citreum; M. Sornthali; Bact. varicosum.
1913- 1 . .	M.	43	2	O	B. subtilis.
1913- 5 . .	F.	56	6	O	O
1913- 6 . .	M.	43	5	O	Cladothrix invulnerabilis.
1913- 8 . .	M.	40	10	O	" "
1913-12 . .	F.	42	18	Diphtheroid bacillus.	Diphtheroid bacillus.
1913-14 . .	M.	39	3	B. beta.	B. Rheni.
1913-20 . .	M.	65	2	Moulds.	B. nitrificans.
1913-22 . .	F.	64	36	B. cloacæ.	M. simplex.
1913-23 . .	M.	49	2	M. simplex.	M. simplex rub.; M. xanthogenicus.
1913-30 . .	M.	45	14	M. carneus; M. endocarditis.	M. polymositis.
1913-31 . .	M.	54	12	Planococcus tetragenus.	M. alvi.

Although Gay and Southard failed to find any diphtheroid organisms in thirteen general paretics, we here find, out of a series of twenty-six general paretics, one case in which a diphtheroid organism was found, both in heart's blood and cerebrospinal fluid.

Table VII. (Gay–Southard) is to be compared with Table VIII. showing fourteen cases in which the cerebrospinal fluid and blood in our series remained sterile. Closer analysis of these probably would cause some suspicion as to 1912–4, terminal disease, bronchopneumonia two days; and 1912–13, general paretic, with bronchopneumonia of two days and bed sore of a month, and 1913–25, bronchopneumonia of three days' duration. The others were very probably actually sterile or at least presented no organisms by the aërobic method.

TABLE VII. (Gay and Southard, 1910.)

Cases with blood and cerebrospinal fluid sterile.

Aut. No.	Sex.	Age.	Hours P.M.	Mental Disease.	Duration of Terminal Disease.
1105 . . .	M.	86	4	Epilepsy; dementia.	10 days.
1136 . . .	M.	46	9	Epilepsy; cerebral glioma, 1 to 2 years.	12 hours.
1139 . . .	F.	77	4	Manic-depressive insanity, 37 years; arteriosclerosis.	2 (possibly 12) days.
1165 . . .	F.	59	144	Dementia precox, 27 years.	2 (possibly 7) days.
1168 . . .	F.	24	78	Dementia precox, 4 years.	8 months.
1179 . . .	M.	72	8	Organic dementia, 4 years.	2 years.
1218 . . .	F.	55	22	Alcoholic dementia, 22 years.	6 years.
1222 . . .	M.	71	1	Dementia, 10 years.	4 days.
1224 . . .	F.	62	168	Epilepsy, 13 + years.	2 days.

TABLE VIII. (1913.)

Cases in which blood and cerebrospinal fluid were sterile.

No.	Sex.	Age.	Hours P.M.	Mental Disease.	Terminal Disease.
1911–38 . .	M.	66	14	Senile dementia, 7 months, 25 days.	Acute ulcerative colitis, 11 days.
1911–47 . .	F.	46	14	Unclassified, 17 months, 14 days.	Pulmonary tuberculosis; intestinal tuberculosis, 1 year, 2 months.
1912–4 . . .	M.	65	4	Arteriosclerotic dementia, 13 months, 2 days.	Bronchopneumonia, 2 days.
1912–12 . .	F.	69	1	Senile dementia, 24 months, 13 days.	Apoplexy; chronic valvular heart disease; bronchopneumonia, 1 day.
1912–13 . .	M.	54	16	General paresis, 10 months, 2 days.	Bronchitis, 2 days. Bedsore, 1 month.
1912–14 . .	M.	45	10	Unclassified, 2 months.	Pulmonary tuberculosis, 2 months plus.
1912–17 . .	M.	23	4	Dementia precox, 20 months, 23 days.	Pulmonary tuberculosis, 2 months plus.
1912–18 . .	M.	72	3	Arteriosclerosis, 26 days.	Ulcerative colitis, 5–7 days.
1912–19 . .	M.	53	7	Involution melancholia, 17 months, 27 days.	Chronic nephritis, unknown duration.
1912–44 . .	M.	76	4	Senile psychosis, duration ? ?	Bronchopneumonia, duration ?
1913–5 . . .	F.	56	6	General paresis, 29 months, 14 days.	Acute symptoms, 1 day.
1913–10 . .	M.	62	1	Arteriosclerosis or trauma, 26 months, 16 days.	Chronic valvular heart disease; unknown duration.
1913–25 . .	F.	51	14	Arteriosclerotic dementia, 21 months, 16 days.	Bronchopneumonia, 3 days.
1913–32 . .	M.	51	18	Arteriosclerotic dementia, 24 months, 1 day.	Arteriosclerotic; convulsions, 1 day.

Histology of the frozen sections of the nervous system in the cases which showed no growth in heart's blood or cerebrospinal fluid — fourteen in number — follows:

1911–38. Section from gyrus rectus. — Faint reaction in nerve cells. The larger vessel walls are the chief point of attack.

1911–47. Shows brilliant fat reaction in all the pyramidal cells and quantities in vessel walls.

1912–4. Section of lumbar cord: no fat (except nerve cell pigment).

1912–12. Brilliant fat staining in all layers of cells in a section from the gyrus rectus, particularly marked in fourth and sixth layers, though

the cells are not sharply marked and the processes are indistinct. The vessel walls show brilliantly red.

1912–14. Some of the nerve cells are much distorted and swollen. These show fat. Others free.

1912–17. No fat seen in the cells. Scattered fat in vessel walls.

1912–19. Sections of lumbar cord: no fat.

1912–18. Frozen sections of the gyrus rectus show few nerve cells in normal size or in normal arrangement. Where the cells have taken up the Sudan-3 stain, it is faint in color, scattered and scanty. Slight in vessel walls.

1912–44. Section of lumbar cord: few flecks in the vessel walls and the periphery of the cord.

1913–5. Gyrus rectus shows in all layers a marked fat deposit in nerve cells; glia cells and vessels share in the change.

1913–10. Unmistakable perivascular infiltration with lymphocyte and plasma cells were destructive of nerve cell lamination and overgrowth of glia cells. Nerve cells are distorted with slight peripheral staining (Sudan-3) (a case of general paresis clinically undiagnosticated).

1912–13. Fat present in all layers of nerve cells to a slight degree. No one of the nerve cells shows it in the processes.

1913–25. Fat in every layer of nerve cells, patchily also in the form of granules on the walls of the vessels surrounding and in the endothelial cells.

1913–32. Lumbar cord: no fat.

To sum up, one case, with onset of symptoms in the third decade (1912–17), dead at twenty-three of dementia precox and pulmonary tuberculosis, quite negative culturally (not only in the heart's blood and cerebrospinal fluid, but also in right middle ear and mesenteric lymph node), showed by the Marchi method no change and none in frozen section by fat stains. The other thirteen cases ranged from forty-six to seventy-two years of age, so that age may perhaps have something to do with the fat reactions of the nerve cells (compare with the series of nine cases which had the same organism in both heart's blood and cerebrospinal fluid).

TABLE IX. (Gay and Southard, 1910.)

The bacteriology of cases which did not show diffuse fatty changes in the spinal white matter.

No.	Sex.	Age.	Hours P.M.	Katamnesis.	Blood.	Cerebrospinal Fluid.
1097 . .	M.	29	14	Phthisis, 1 + years.	Bact. ambiguum.	M. albus; M. aureus; unidentified bacillus.
1099 . .	M.	67	13	Ileocolitis, 1 week.	O	B. proteus vulgaris; B. proteus zenkeri.
1113 . .	F.	64	25	Diarrhea, 16 days.	M. albus.	M. albus.
1123 . .	F.	28	6	Endocarditis, 9 + days.	O	" "
1134 . .	F.	66	3	Bronchopneumonia, duration brief.	B. fuscus.	B. coli, 25 col.
1142 . .	M.	81	7	Pneumonia and retraction of head, several days.	M. aureus.	Contaminated.
1144 . .	M.	69	26	Lobar pneumonia, 2 days.	Strep. albus.	B. coli, 1 col.
1145 .	F.	37	22	Exhaustion, months.	M. cereus; M. nivalis.	O
1148 . .	F.	25	4	Acute symptoms, 2 days.	O	M. albus.
1155 . .	M.	63	1	Bronchopneumonia, duration?	O	M. citreus.

TABLE X. (1913.)

The bacteriology of cases which did not show diffuse fatty changes in the spinal white matter.

No.	Sex.	Age.	Hours P.M.	Katamnesis.	Blood.	Cerebrospinal Fluid.
1911.2	F.	78	9	Acute hemorrhagic colitis; multiple thrombi.	M. cremoides.	M. rubescens.
1911.3	F.	57	10	Brain tumor.	O	M. concentricus.
1911.8	F.	48	15	General paresis.	Sarc. pulmonum.	M. pyogenes.
1911.9	M.	73	7	Bronchopneumonia.	O	M. cereus.
1911.10	M.	66	2	Exhaustion.	O	Strep. pyogenes.
1911.16	M.	57	5	General paresis.	Bact. influenzæ.	B. Wesenbergii.
1911.17	M.	77	45	Bronchopneumonia.	O	Bact. anthracoides.
1911.20	F.	53	12	Bronchopneumonia; diabetes.	O	M. carneus; Bact. gracile.
1911.33	M.	79	8	Cystitis.	O	
1911.34	F.	52	16	Bronchopneumonia.	O	Bact. anthracoides.
1911.35	F.	59	3½ days.	"	O	M. xanthrogenicus; Bact. gallinarum.
1911.39	M.	54	15	Bronchopneumonia; acute cystitis.	O	Unidentified coccus.
1912.7	M.	42	68½	Pulmonary tuberculosis.	M. cumulatus.	M. ovalis.
1912.17	M.	23	4	Pulmonary tuberculosis.	O	O
1912.18	M.	72	2½	Cerebral arteriosclerosis.	O	—
1912.19	M.	53	7	Chronic nephritis.	O	
1912.20	M.	54	18	General paresis.	O	Contaminated.
1912.53	M.	52	19	" "	Bact. truncatum.	M. expositionis.
1913.54	M.	46	3	Bronchopneumonia.	O	Bact. limbatum.
1913.18	F.	73	8	Acute vegetative endocarditis; acute fibrinous pericarditis; chronic interstitial nephritis.	M. candicans.	B. erodens.

TABLE XI. (Gay and Southard, 1910.)

The bacteriology in cases of terminal disease with assigned duration of four days or less.

No.	Sex.	Age.	Hours P.M.	Katamnesis.	Blood.	Cerebrospinal Fluid.
1084	F.	46	9	Convulsions, 4 days.	Contaminated.	Micrococci, unidentified.
1090	F.	57	45	" 2 days.	B. coli; B. flavus.	B. coli.
1103	F.	71	96	Bronchopneumonia, 3 days.	Streptococcus.	Streptococcus; B. pyocyaneus; unidentified diplobacillus.
1104	M.	50	23	Occlusion of larynx.	O	M. albus and aureus.
1110	M.	60	3	Otitis media, 2 + days.	Contaminated.	M. citreus.
1114	F.	79	26	Fever, 3 days.	M. albus.	M. albus.
1117	M.	73	3	Pneumonia, 4 + days.	M. aurantiacus.	Contaminated.
1130	F.	56	12	Fever, a few days.	B. coli; M. aërogenes.	O
1135	F.	28	8	Pneumonia, 3 days.	O	M. concentricus.
1136	M.	46	9	Convulsions, 12 hours.	O	O
1139	F.	77	4	Bronchopneumonia, 2 days.	O	O
1140	M.	63	16	Cerebral hemorrhage, 2 days.	M. albus.	Contaminated.
1142	M.	81	7	Recent pneumonia, with retraction of head.	M. aureus.	"
1144	M.	69	26	Lobar pneumonia, 2 days.	Streptococcus; M. albus.	B. coli.
1148	F.	25	4	Acute symptoms, 2 days.	O	M. albus.
1149	M.	57	4	Perforation of peritoneum, 2 days.	B. coli.	O
1150	M.	78	4	Recent pneumonia.	O	B. coli.
1151	F.	38	4	Pneumonia and colitis, 2 days.	Pneumococcus.	M. citreus.
1159	F.	68	20	Rupture of heart.	O	B. coli.
1165	F.	59	144	Fainting spell.	O	O
1166	F.	61	17	Coma, 2 days.	M. cereus.	B. coli.
1173	F.	65	1	Fever a few days.	M. aurantiacus.	" "
1174	F.	48	45	Hemorrhagic pancreatitis, 3 days.	B. coli.	" "

TABLE XI.— *Continued.*

No.	Sex.	Age.	Hours P.M.	Katamnesis.	Blood.	Cerebrospinal Fluid.
1178 . .	M.	71	27	Lobar pneumonia, 2 days.	M. albus.	O
1181 . .	F.	77	6	Bronchopneumonia.	Streptococcus.	
1182 . .	F.	72	69	Vomiting and weakness, 2 days.	O	M. albus; unidentified bacillus.
1184 .	F.	42	15	Exhaustion, a few days.	M. albus.	B. coli.
1186 . .	F.	33	22	Purpura hemorrhagica, 3 days.	" "	M. albus.
1214 . .	M.	66	12	Bronchopneumonia, recent.	B. coli.	O
1222 . .	M.	70	1	Bronchopneumonia, 4 days.	O	O
1224 . .	F.	63	168	Acute symptoms, 2 days.	O	O

TABLE XII. (1913.)

The bacteriology in cases of terminal disease with assigned duration of four days or less.

No.	Sex.	Age.	Hours P.M.	Hours Room Temp.	Katamnesis.	Blood.	Cerebrospinal Fluid.
1911.1	F.	66	2	½	Bronchopneumonia.	O	B. aërogenes.
1911.4	F.	65	15	½	"	Planococcus tetragenus.	B. unidentified.
1911.6	M.	80	34	15	"	M. xanthogenicus.	M. xanthogenicus.
1911.9	M.	73	7	5	"	O	M. cerasinus.
1911.12	M.	57	14	13	Ulcer esophagus.	O	—
1911.17	M.	77	45	15	Bronchopneumonia.	O	Bact. anthrocoides.
1911.19	F.	67	7	4	"	M. pyogenes.	M. cremoides; Bact. sanguinis.
1911.20	F.	53	12	½	"	O	Bact. alvei.
1911.21	F.	78	1	½	"	O	M. ovalis.
1911.22	M.	73	9	5	Lobar pneumonia.	O	M. cereus.
1911.24	F.	72	12	½	Bronchopneumonia.	O	M. cereus.
1911.28	F.	42	2	½	"	Bact. alvei.	M. pyogenes.
1911.30	F.	44	1	½	Perforated gastric ulcer.	O	Strep. carnis.
1911.34	F.	52	16	½	Bronchopneumonia.	O	M. xanthogenicus; Bact. gallinarum.
1911.35	F.	59	3½ days.	½	"	O	M. xanthogenicus; Bact. gallinarum.
1911.36	M.	67	3	½	Hemorrhagic colitis.	M. carneus; M. cereus; Bact. schottelii.	Bact. schottelii; Bact. limbatum.
1911.39	M.	54	15	14	Bronchopneumonia.	O	Coccus unidentified.
1912.1	F.	78	9	5	"	Bact. gallinarum; M. xanthogenicus; M. coccus aurantiacus.	—
1912.2	F.	73	7	½	Cyst in bulb.	Bact. limbatum.	Bact. limbatum; Bact. schottelii.
1912.4	M.	65	4	3	Bronchopneumonia.	O	O
1912.8	F.	53	6	3	Ruptured gastric ulcer.	M. beta.	M. beta.
1912.11	M.	84	6	5	Acute dilatation heart.	M. cremoides.	O
1912.12	F.	69	1	½	Bronchopneumonia.	O	O
1912.16	M.	72	16	15	"	Bact. mycoides.	O
1912.42	M.	36	3	2	"	Staphylococcus.	O
1912.44	M.	76	4	3	"	O	O
1912.46	M.	59	14	12	"	B. vulgatus.	O

TABLE XII. — *Continued.*

No.	Sex.	Age.	Hours P.M.	Hours Room Temp.	Katamnesis.	Blood.	Cerebrospinal Fluid.
1912.48	M.	39	10	9	Bronchopneumonia.	B. leodermos.	B. murisepticus.
1912.50	F.	52	15	14	Acute hemorrhagic pancreatitis.	O	B. murisepticus; M. stellatus.
1912.54	M.	46	3	2	Bronchopneumonia.	O	Bact. limbatum.
1913.2	M.	81	3	2	Acute hemorrhagic pachymeningitis.	B. subtilis.	M. nivalis.
1913.3	F.	80	3	2	Septic cellulitis.	B. coccineus.	B. coccineus.
1913.5	F.	56	6	4	Sudden death.	O	O
1913.6	M.	43	5	4	" "	O	Cladothrix invulnerabilis.
1913.7	F.	64	2	1	Bronchopneumonia.	Strep. acidilactici.	B. cinctus.
1913.8	M.	40	10	8	Lobar pneumonia.	O	Cladothrix invulnerabilis.
1913.12	F.	42	18	17	Convulsive movements.	Diphtheroid.	Diphtheroid.
1913.13	F.	81	15	14	Bronchopneumonia.	Contaminated.	B. fermentationis.
1913.14	M.	39	3	2	"	B. beta.	B. rheni.
1913.15	F.	52	20	½	Ruptured aneurysms.	Contaminated.	Contaminated.
1913.17	F.	83	2	1½	Bronchopneumonia.	Streptothrix invulnerabilis.	Planococcus citreus; B. intestinalis; B. fissuratus.
1913.18	F.	72	8	7	Acute fibrinous pleuritis.	M. candicans.	B. erodens.
1913.21	F.	83	11	10	Lobar pneumonia.	Contaminated.	—
1913.25	F.	51	14	13	Bronchopneumonia.	O	O
1913.28	M.	65	2	1½	Thrombus aorta.	M. aethebius.	B. vulgatus.
1913.33	M.	38	27	15	Bronchopneumonia.	Bact. viscosum.	B. murisepticus.

TABLE XIII.

Nine cases with same organisms in cerebrospinal fluid and heart blood.

1911–6	M. xanthogenicus.	Pathogenic for small birds.
1911–23.....	B. cloacæ.	Non-pathogenic.
1911–32.....	M. xanthogenicus.	M. rubescens, the latter not pathogenic.
1911–37.....	Bact. schottelii.	Pathogenicity not established.
1912–2	Bact. limbatum.	" " "
1912–8	M. beta.	Pathogenic for white rats.
1913–3	B. coccineus.	Pathogenicity not established.
1913–12.....	Diphtheroid.*	
1913–34.....	M. salivarius.	Pathogenic for laboratory animals.

* Ford Robertson's bacillus?

Forty-six cases proved to belong in a group which we arbitrarily define as clinically negative. By clinically negative for the purposes of this investigation we mean cases with a terminal disease of less than four days' duration. This definition was adopted by Gay and Southard because the prevalent idea is that it takes from three to five days to produce visible fatty degeneration of Wallerian type, *e.g.*, in the distal portion of a sectioned nerve fiber. Four days was thought to be a fair interval, then, for the development of other types of fatty degeneration.

Parenthetically, it must be granted that visible fatty degeneration can probably be produced in less time than four days. In fact, Gay and Southard themselves offered data in their work on anaphylaxis showing that visible fatty change may possibly even be peracute in its production.

However, granting the likelihood that under ordinary circumstances a period of some days is necessary for the advance of fatty degeneration to a microscopically obvious state, it seems safe to say that nothing striking might be supposed to develop as a result of bacterial action, if the bacteria had not been at work some four days prior to the death of

the subject. Analyzed from this point of view, the cases that died suddenly or after exceedingly brief last illnesses actually do prove to show far less evidence of diffuse fatty change than do the cases with last illnesses of more chronic character. Indirectly, this evidence might be used to support the contention that some of the bacteria found in the long-duration cases may have caused the fatty changes more common in these long-duration cases.

In nine cases the same organism was found in the cerebrospinal fluid and heart's blood, four of them being organisms which are recognized as pathogenic (Chester). The histology of frozen section with the Herxheimer method of fat staining showed:

In 1911–6, a large amount of fat in all cell layers of cortex (gyrus rectus), particularly the internal and large pyramids.

1911–23. Frozen sections from the lumbar cord showed no changes, except that there was fat in the lumina of certain capillaries and pigment in most of the ganglion cells (not extending into the processes of these cells).

1911–32. Frozen sections from the cerebellum showed fat in the lumina and endothelium of vessels and in some of the multipolar nerve cells (sometimes stated to be rarely affected).

1911–37 Frozen sections of the lumbar cord show fat in the cells and meninges of posterior root zone and slightly in the vessel walls. In the gray matter the ganglion cells retain their shape fairly well but are crowded with fat granules. Some occupy all the space of protoplasm; others only at the fundus. Scattered and clumped fat droplets surround the cells of the ependyma in the central canal and in the neuroglia cells in the white matter.

1912–2. Fat stains show nerve cells of all layers (gyrus rectus) full of fat, and vessels also crowded with it; also in the endothelial cells.

1912–8. Endothelial cells of the vessels show much fat. The third, fourth, and sixth layers (gyrus rectus) show fat in the cells, which extends into the processes, and the glia cells in the seventh layer and in the white matter show granules at their periphery.

1913–3. Gyrus rectus shows brilliant fat staining in the nerve cells and in the vessel walls.

1913–12. Shows brilliant fat reaction in the nerve cells.

1913–34. Gyrus rectus shows diffuse fat in the cells, free layer of the cortex and in the vessels, especially capillaries, and in the third, fourth, and sixth layers of the nerve cells.

SUMMARY AND CONCLUSIONS.

(N.B. — The conclusions have been numbered to correspond with the conclusions of Gay and Southard, 1910.)

1. In a study similar in scope to that of Gay and Southard, 1910, the writers present the results of a second series of one hundred bacterial cultivations from the heart's blood and the cerebrospinal fluid post-mortem in cases of mental disease. The new series is from the Boston, instead of the Danvers, State Hospital.

2. The bacteria were cultivated upon agar plates inoculated with 1.5 cubic centimeters heart's blood and others with 1.5 cubic centimeters cerebrospinal fluid. The cerebrospinal fluid was removed from the third ventricle through the infundibulum, severed at its origin.

3. Forty-four per cent of the blood cultures remained sterile (Gradwohl, 22 per cent; Gay and Southard, 41 per cent; Otten, 42 per cent; Simmonds, 48 per cent).

4. Twenty-four per cent of the cerebrospinal fluid cultures remained sterile (Tomlinson, 28 per cent; Gay and Southard, 28 per cent).

5. Under somewhat different laboratory conditions, the Boston laboratory (bodies often not ice-cooled and always held at a somewhat higher temperature than at Danvers) thus paradoxically yields more steriles than the Danvers laboratory in the blood (44 : 41) but less in the cerebrospinal fluid (24 : 28).

6. It is interesting, however, that the Boston series shows fewer cerebrospinal positives in the earlier than in the later periods post-mortem: a tendency quite the reverse to that of the Danvers series, where the icing (often freezing) of the cadavers may well have inhibited the growth of bacteria post-mortem

7. On the other hand, the heart's blood results are not readily interpretable on the above or any other basis, unless we invoke special bactericidal properties in the sera of different cases.

8. With the lapse of time post-mortem, accordingly, the cerebrospinal fluid certainly seems to show the effect of its

non-bacteriolytic properties (see conclusions 7–9 of Gay and Southard's article quoted above), at a comparatively early date, in the increased frequency of its positives when temperature permits (Boston) as against no increased frequency (or reduction?) when temperature is unfavorable (Danvers).

9. The heart's blood with its (for some time) persistent bacteriolytic substances does not seem to show notable variation in its incidence of positives as time post-mortem elapses under either Boston or Danvers conditions.

10. As to particular bacterial forms, cocci prevail in both series, and in both series there were more cultivations of cocci from the cerebrospinal fluid than from the blood.

11. At Danvers no diphtheroid organisms were picked up in bodies of paretics (*pace* Ford Robertson, 1906); at Boston there was one such instance from the blood and fluid of a case. This as well as other considerations concerning secondary invasions in general paresis will be taken up in a separate communication.

12. Cultivations from twenty-six general paretics are listed; cocci no longer especially prevail in the positive cases (contra Danvers).

13. No conclusion as to the possible relation of bacterial invaders to fatty changes can be drawn from the sterile cases, since all the cases but one were too old to be free from the suspicion of age-changes in the production of lipoid alterations; there was, however, one case, twenty-three years of age, probably quite sterile throughout, and this case showed no lipoid alterations in any part studied.

14. On the other hand, there were twenty cases in which no fatty changes were found histologically and of these no case showed Bacillus coli communis, and cocci prevail as in the Danvers series. Thus, whether we assume various lipoid alterations to go on ante-mortem or post-mortem, and under the influence of bacteria or not under such influence, it would appear that cocci can hardly be charged with effecting such lipoid changes.

15. Forty-six cases are listed, having had symptoms less than four days in duration — as against thirty-one in the

Danvers series; eighteen per cent of the new series, examined by fat-staining methods, proved negative as against twenty-nine per cent histologically negative in the Danvers series; and the eighteen per cent thus both clinically and histologically negative in the Boston series contrast with fourteen per cent histologically negative in the total Boston series — a variation in the same direction as in the Danvers series (29 per cent: 10 per cent) only less striking.

16. In eight cases chosen as showing most marked fatty changes, there were no instances of colibacillosis (one case only of anaërogenes bacillemia); in fact five cases were negative in the cerebrospinal fluid, and six negative in blood.

17. One of the highly (Marchi) degenerated cases (among the eight just mentioned) yielded Cladothrix invulnerabilis in the cerebrospinal fluid; a second case with the same finding also showed degeneration, though of less marked degree; two of the highly degenerated cases (of the eight *supra*) yielded Bacillus murisepticus; another case showed degenerations but less marked.

18. There were nine cases of generalized softening of brain tissues in the Boston series as opposed to thirteen in the Danvers series; moreover, three of these Boston cases were autopsied from three to eight and one-half days post-mortem (at a time when ferment action may be presumed to be under way); no case showed Bacillus coli communis; the bacteria found were in two instances reputed pathogens (Bacterium varicosum and Micrococcus salivarius); in three other cases the organisms were either liquefiers of various media (Bacillus subtilis, Micrococcus alvi) or abstractors of water (Cladothrix invulnerabilis); in only one case was the bacteriology negative (a case of ulcerative colitis).

19 Only indirect evidence concerning the effect of Bacillus coli communis or its toxines on nerve fiber degeneration is here afforded; however, the organisms which are associated with the soft brains in the Boston series are in most cases also liquefiers of various laboratory media.

Epicritical conclusions. — The conclusions of the new study in general coincide with those of Gay and Southard, but present some novel points (especially conclusions 6, 8, 14, 16, 18), and these points may be briefly considered as follows:

20. It is suggested by a comparison of the two series that the more immediate and thorough cooling (or even icing) of the Danvers cadavers has served to inhibit the growth of bacteria in the cerebrospinal fluid, since the lapse of time post-mortem is attended in the Boston series by increasing frequency of positives in the cerebrospinal fluid.

21. The blood findings show no such effects as those just mentioned for the cerebrospinal fluid; perhaps the curve is spoiled by the presence or absence in special cases of bactericidal substances in the blood.

22. Whatever may be said of the possible ante- or post-mortem effects of other organisms in producing lipoid changes in the nervous system, the cocci as a group may be absolved from this charge; if the cocci act at all ante-mortem, it must be rather in the direction of irritative than of destructive effects.

23. The absence of colibacillosis in the Boston series is striking; a few other organisms, either pathogenic or somewhat destructive to the laboratory media used for their cultivation, enter to take the place of Bacillus coli communis in the "soft brain" cases.

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XLV

ON THE TOPOGRAPHICAL DISTRIBUTION OF CORTEX LESIONS AND ANOMALIES IN DEMENTIA PRÆCOX, WITH SOME ACCOUNT OF THEIR FUNCTIONAL SIGNIFICANCE.*

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CONTENTS.

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* Being contributions from the Massachusetts State Board of Insanity Number 25 (1914.5) and Danvers State Hospital Contributions Number 53. The substance of this contribution was presented at a meeting of the American Neurological Association held at the Triennial Congress of Physicians and Surgeons, Washington, D. C., May, 1913. (*Bibliographical Note.* —The previous S. B. I. Contribution Number 23 (1914.3) was by E. E. Southard and M. M. Canavan, entitled "The Significance of Bacteria Cultivated from the Human Cadaver: A Second Series of 100 Cases of Mental Disease with Blood and Cerebrospinal Fluid Cultures and Clinical and Histopathological Correlations," to be shortly published in Journal of Medical Research.)

I. Introduction.

I shall not here insist on the fundamental importance of deciding how far dementia præcox is a structural disease and how far functional. Nor shall I repeat what I have said before concerning the partisan attitude adopted by many workers in this controversial field.[1] A portion of the difficulty is beyond question due to the fact that, amid the cloud of witnesses, many speak who have no clear conception of what the terms structure and function mean.[2,3] But, brushing these writers aside, I am inclined to think that many persons who could offer a reasonable account of what they mean by the term function are treating the dementia præcox problem, not so much on its own merits, as on the supposed merits of some general conception of the nature of mental disease. Thus, if A believes that "*insanity is brain-disease,*" B that "*insanity is disease of function,*" C that "*insanity is faulty adaptation of the individual to the environment,*" D that "*insanity is hereditary,*" E that "*insanity is due to the operation of subconscious 'complexes,'*" the chances are against the preservation by A, B, C, D, or E of a non-partisan attitude to the particular problems of dementia præcox (to say nothing of prejudice to the entire problem of mental disease). So far as I can see, each of the propositions, A, B, C, D, E, and many others might be entirely true, and still consistent with the kind of contentions which I made in 1910 in my paper entitled, "A Study of the Dementia Præcox Group in the Light of Certain Cases Showing Anomalies or Sclerosis in Particular Brain Regions."

Nevertheless I assume that many readers will desire to know what particular form of prejudice I myself secretly entertain concerning mental disease! Without betraying myself to the uttermost depths and executing a veritable "catharsis" (*pace* Freud) of logical conceptions, let me state that for the most part I do not allow myself to admit entertaining any prejudice in this regard. Positive as were the findings in 1910, and harmonious as they were with the conceptions of Nissl, Alzheimer, and, more generally speaking, of Kraepelin, the findings came as a surprise to me, since my preconception had been, if anything, that the brain would be found essentially normal in dementia præcox, *i. e.*, an example of the concept of "*discords played on good instruments.*" As a result of actual investigation, however, it seemed probable that

dementia præcox brains, at least at autopsy, no longer looked like "good instruments," and I was forced to illustrate the concept of discords on good instruments from the field perhaps of manic-depressive insanity. This latter problem I am studying at present with methods similar to those adopted for dementia præcox in 1910.

But, granting the occurrence of lesions or anomalies in dementia præcox brains such as those described, I had to face the contention of Adolf Meyer[4] that the disease is possibly but "*incidentally organic.*" And, even if the disease is proved to be essentially—rather than incidentally—organic, the question was outstanding whether the described changes were instances of cerebral *agenesia* (as I understand August Hoch to maintain), or whether they are also instances of *acquired* lesion (*aplasia or hypoplasia,* rather than agenesia). This latter question I had specifically taken up in my paper of 1910, and I then found over half of my cases to show "congenital" features regarded as hypoplastic. The vast majority of the cases seemed to me to show evidences of "acquired" change.

In this state of the problem it seemed imperative to do more work of the sort initiated in 1910.

In 1910 I was able (a) to show that a vast majority of cases of dementia præcox is characterized by *coarse anomalies or scleroses in particular regions of the cerebral cortex.* I went on (b) to draw certain tentative conclusions as to structure and function which seemed to be borne out by those common clinical features, *e. g., paranoia* and *catatonia,* found in *cases having lesions predominant in the frontal and the parietal regions, respectively.* Now in 1914 I am able to substantiate the former claim and, on the basis of systematic brain photography, to carry out a more thorough convolutional analysis than has been hitherto attempted. It seems to me that I am thus able more safely to attack the moot question of *congenital versus acquired features* in the genesis of this disease or the diseases of this group. As to the latter claim (clinical correlations) I have been greatly aided by the appearance, in my *new series,* of cases showing anomalies or other *lesions of the temporal convolutions.* These new cases, for some reason only sparingly exemplified in the 1910 series, give rise to interesting speculations as to the part played by the *thought-speech-action* mechanism in dementia præcox.

The new series (1914) consists of 25 cases, 22 of which were not available in the 1910 series of 28 cases. Since three cases appear in both series (having now been systematically photographed and analyzed as to convolutions), the more general conclusions which can be drawn at this stage of the investigation relate to a total series of 50 cases. Of about 45 of these cases I believe we are entitled to say that their brains showed gross anomalies or other lesions of an important character.

Before rehearsing the nature of these appearances, a preliminary question must be answered. What is the *principle of selection* of these cases? Granting that dementia præcox is an entity or a group of entities, what title have these particular cases to the diagnosis? In the first place, I have given clinical histories, which, though condensed, are sufficient to permit the reader to make up his own mind as to the probable diagnosis. In the second place, I have guarded against too high a percentage of gross lesions by excluding a large number of (to my mind) perfectly good cases simply because they showed gross lesions not ordinarily thought to be related with dementia præcox phenomena. This process of exclusion was particularly resorted to in the 1910 series, when such features as *generalized brain atrophy* (five cases all of 11 or more years' duration), *cysts of softening, marked sclerosis of the vessels of the circle of Willis, diffuse chronic leptomeningitis* (11 cases) were regarded as complicating features that should not be considered in a first study of the anatomy of dementia præcox. From my present standpoint I am by no means so sure that generalized brain atrophy or even chronic diffuse leptomeningitis may not play a significant part in some cases or sets of cases in the total group. Accordingly in the 1914 series, when it was planned to publish the brain photographs themselves for the reader's judgment, not so much attention has been paid to excluding cases. In fact, the present collection represents a perfectly fair sample of dementia præcox cases as available in a large hospital for the insane (Danvers State Hospital), which has not only the features of a nursing asylum (in the European sense), but also the receiving function for a large and fairly representative district (Essex County, Massachusetts).

The series of cases is therefore a random non-selected series from which I have particularly prevented the more normal-looking

brains from being lost, inasmuch as normal-looking brains in the insane have been a special object of attention in my more recent work.

I therefore assume that, granting dementia præcox to be an entity, this series belongs both clinically and anatomically to a fair sample thereof; if not, the appropriate cases can be readily excluded. *If, however, dementia præcox is not an entity,* then I must fall back on the fact that in any event these cases are all cases of some sort of mental disease having pretty clearly (as a rule) adolescent or early adult onset. If we are not studying dementia præcox because it is non-existent, then at least we are studying mental disease. I have myself no suspicion that the *unity of insanity* will ever get much standing again in any important sense of the term *unity*. But, of course, fresh etiological work in the future will surely carve out new entities from some of our material. Our hope is to gain, by work on the processes of pathogenesis, ideas for prosecuting work in etiology. The most concrete suspicion which might attach to this material is the fear that some other current entity (*e. g.,* manic-depressive insanity) is masquerading in our series as dementia præcox. As to this I would say that the inclusion of such cases should theoretically serve only to lower our percentage of lesion-cases; also, that material has been collected and is being analyzed on similar lines for precisely the problem of manic-depressive insanity.

I do not need to describe more especially at this point the nature of the the lesions found in 1910, inasmuch as the present series will amply illustrate them. I will, however, here reproduce paragraphs 9 to 17 of the 1910 conclusions in order to show the status of the problem at that time.

9. Palpable glioses of a focal or variable character, combined in numerous instances with visible atrophy and microgyria have been found in over half the series under examination, in cases regarded as clinically above reproach and *not* subject to coarse wasting processes, focal encephalomalacia, cortical arteriosclerosis, or diffuse chronic pial changes.

10. The frequent co-existence of several foci of sclerosis or atrophy in the same brain, and the microscopic examination of milder degrees of nerve-cell disorder and gliosis in regions without gross lesions tend to the conception that the agent is more general and diffuse in its action than would seem at first sight, so that future research may well demonstrate that certain instances of coarse brain wasting, and even of diffuse chronic leptomeningitis, belong in the group (microscopic corroboration necessary for assigning values to focal variations).

11. The microscopic examination of the residue of cases in which gross lesions or anomalies were not described shows the same tendency to gliosis and satellitosis in numerous instances, and the same tendency to focal variations from gyrus to gyrus exhibited by the gross lesion group. These findings suggest that the minor gross lesions and anomalies of several cases actually escaped notice (the protocols, though drawn up with a certain system are by various hands) at autopsy, so that the probable actual proportions of gross lesions is 68 per cent. If microscopic evidence is resorted to, the "organic" proportion in our series rises to 86 per cent.

12. Several groups of cases were classified from the distribution of macroscopic lesions, although the focal purity of these cases can often be brought in question from the results of microscopic examination (infrastellate gliosis and satellitosis also in macroscopically "normal" areas).

I. Pre-Rolandic group, including a superior frontal-prefrontal sub-group of paranoidal trend (*cf., e. g.,* case 1061).

II. Post-Rolandic group, including (a) postcentral-superior-parietal (sensory perceptual) sub-group in which katatonic features are the common factors (*cf., e. g.,* case 1298); (b) occipital sub-group (*cf.,* case 1149).

III. Infra-sylvian group (too small for clinical correlations).

IV. Cerebellar group (katatonic features).

13. If these data find general confirmation they will doubtless go far to unify discussion, since mild, variable and progressive intracortical lesions, proceeding at different rates in different parts of the apparatus, and having the peculiar distributions indicated above, would explain adequately some of the contentions of the dissociationists, while remaining not wholly inconsistent with Kraepelinian ideas.

14. The frontal-paranoid correlation is in line with modern physiological ideas, but it must be granted that the occipital and temporal regions, as elaborating important long-distance impulses, may well play a part also in paranoid states.

15. The cerebellar-catatonic correlation is doubtless in line with some contentions of the Wernicke school, and obvious comments might be made in connection with the proprioceptive functions of the cerebellum (Sherrington).

16. The postcentral-superior-parietal relations to catatonic symptoms are perhaps theoretically the most novel suggestion from the work, but here again the results are not inconsistent with modern physiology.

17. The topographic study of dementia præcox brains, both gross and microscopic, is commended as likely to shed new light on the pathogenesis of certain symptoms, notably paranoidal and catatonic symptoms.

Since 1910, however, the problems of dementia præcox have shifted somewhat, and clearer views of the schism as to genesis which was then raging have now been rendered, more especially

by Bleuler and by Kraepelin himself. Let us first listen to the more recent conclusions of Kraepelin as to causes, especially those operating from without.

II. Some Recent Views of Dementia Præcox (Kraepelin, Bleuler, Meyer)

On account of the Kraepelinian view of dementia præcox as in some sense an organic disease, the new edition,[5] volume III, 1913, was eagerly looked forward to by all workers, and the more because the Kraepelinian view had been met by the careful and ingenious volume on Dementia Præcox or the Group of Schizophrenias (published in Aschaffenburg's Handbuch) by Bleuler, 1911.[6]

Among causes, Kraepelin considers age, sex, conditions of life, heredity, germ-plasm disease, idiosyncrasy, which need not especially concern us here. Among external causes *mental strain* (Kahlbaum, Deny and Roy) is first mentioned, only to be dismissed because abounding evidence of dementia præcox exists in persons and races not subject to such strain. Next, *infections during development* (Vigouroux and Naudascher, Bleuler) are considered, but the low percentage of initial infections (10-11% in Heidelberg material) and the difficulty of assuming a progressive effect wrought by such infectious agents as those of typhoid fever or scarlet fever (the most frequent initial infections) or the short streptococcus of Bruce militate against the infection hypothesis. Too great rarity holds also of the however not infrequent syphilis (Steiner and Pötzl), head injuries (Muralt), brain tumor, alcoholism.

Concerning these dismissed causes, I assume that Kraepelin would be the first to admit that their dismissal depends on admitting the diseased dementia præcox to be a unit. I find it hard to deny that, since everyone admits (Kraepelin also) that catatonic symptoms may be produced by *e. g.*, brain tumor, so also might the remains of small destructive cerebral hemorrhages in typhoid fever and in coccal subinfections after scarlet fever, to say nothing of properly situated luetic ravages be able to produce catatonic or even other symptoms of dementia præcox. Nor can our logic be entirely overwhelmed by the progressive nature of "true" dementia præcox, since on the one hand many cases of dementia

præcox are very slightly if at all progressive after the first attack, and on the other hand the reparative and compensating phenomena following critical infectious disease of brain or meninges are sometimes so slow as to be diagnosticated " active " for comparatively long periods.

Accordingly, in a particular case, I believe we should not lightly throw aside the infection hypothesis, even though the major portion of the dementia præcox group can hardly be explained as the result of any known kind of infectious agent.

Again, Kraepelin has little faith in prison conditions (confinement, food, limitation of movement, poor air and light, masturbation) as producers of dementia præcox. On the other hand, he believes that it was the dementia præcox which brought these persons to prison, and not *vice versa.* So also with *prostitution.*

Kraepelin has been far more impressed by *sex factors* as possible causes. The frequency of menstrual disorders in women and the unfavorable effect of menstruation upon the established disease, as well as the not infrequent onset in pregnancy, in the puerperium, or after abortion, serve to call attention to the sex factor in women.

In fact, the subjects of dementia præcox are very frequently *hypersexual* (purposeless masturbation, excesses, and ideas of sexual influences). Masturbation is so frequent and obstinate, especially in men, that the hebephrenic type of the disease has been sometimes known as insanity of masturbation. The disease also not infrequently sets in after the frustration of some plan of marriage. Suppression or faulty development of sexual activity (Tschisch), disturbances in the internal secretions of the sex-glands (Lomer), similarities between catatonic patients and thyroidectomized dogs (Blum), relationship with myotonia, myoclonia, tetany (Lundborg), veratrin-like reaction of muscles (Ajello), headaches reminding one of blood and metabolic disorder (Tomaschny), the occurrence of osteomalacia in dementia præcox (Barbo and Haberkandt), are mentioned by Kraepelin from the literature. Kraepelin thinks, too, that the sexual factors above mentioned, the thyroid symptoms sometimes seen, the tremendous variations in body weight, the occasional hypothermia, the epileptiform attacks, the occasional sudden deaths, all point to some poison circulating in the body. Dementia præcox would thus be a kind of autointoxication due to metabolic disorder.

According to Kraepelin, dementia præcox is more allied to epilepsy, diabetes, gout, chlorosis, than to manic-depressive insanity, hysteria, and psychopathic states.

If one should inquire what might be the source of this dementia præcox autotoxine, Kraepelin has at present no definite answer. Should one think of internal secretions brought out by emotion (like adrenalin, *e. g.,* in recent experimental work with emotion in lower animals), I judge that Kraepelin would not be inclined to entertain such an idea strongly. For, in his critique of Jung, who, he says, thinks that the emotion-laden Freudian complex acts like a trauma or an infection, Kraepelin remarks that, if emotion is to produce toxines, then manic-depressives should be especially prone to dementing intoxication!

Still, as I read recent literature on internal secretions, the intercurrent production of toxines or of toxic agents can hardly be excluded in dementia præcox. Kraepelin has himself accumulated so much (albeit scattering) evidence of sex-factors in dementia præcox that the internal secretory hypothesis can hardly be neglected. Whether such toxic agents are competent to produce dementia or whether their results are, as the logicians say, *reversible,* depends, it would seem, on the toxine.

Contrary to my own conclusions of 1910, and again to the conclusions of the present communication, Kraepelin sums up the status of *dementia præcox anatomy* by stating that there are no coarse brain changes, except now and then pial thickening or pial edema (the latter of agonal origin). As a matter of fact, coarse brain changes are not at all infrequently found even in routine examinations; and in published descriptions of dementia præcox autopsies such coarse changes are often mentioned, although not often emphasized.

The fact is that so much attention has latterly been paid to microscopic changes that the larger issue of the registration of serious destructive changes in the gross has fallen out of view. I cannot understand how pathologists can avoid seeing that the well-marked microscopic changes which they (following Alzheimer and Nissl) so frequently describe must in the nature of things be registered in the gross. Those, however, who are aware how rarely the conditions in neuropathological laboratories are in every way favorable for research (complete control of both head and trunk material

from autopsy table to stained section, opportunity for examination and especially for palpation of the fresh brain, photography of brains from the broadest anthropological viewpoints, ample histological material prepared by various methods with adequate technique, etc.) will not wonder that much has to be neglected in routine work.

But, though Kraepelin finds no reason to take account of gross brain changes, he does refer to numerous workers whose descriptions show *topical variations in the degree of the processes* seen (Mondio, Zalplachta, Agostini, DeBuck and Deroubaix, Dunton, Wada). From their work is concluded that the frontal and central regions, as well as the temporal, are more likely to suffer than the occipital regions.

Moreover, although quite unaware of the anatomical changes described by me in 1910, Kraepelin pushes his interpretation of the topical variations of *microscopic processes* just mentioned to a considerable length, and to some degree on similar lines to those I followed in 1910.

The marked involvement of the frontal region is correlated by Kraepelin with the clinical involvement of the higher intellectual functions as opposed to memory and acquired faculties, not clinically involved.

The fine disturbances which Kraepelin believes, from his analysis of the literature, characterize the *vicinity* of the precentral gyri are correlated by him with the disorders of will and of motility, even perhaps with finer asynergy of muscles. Of course my own analysis gives rise rather to the hypothesis that *post*-Rolandic disorder, presumably affecting kinaesthesia, is more often responsible for catatonia; yet, as in epilepsy, so in dementia præcox, disease approaching very near the projection system may now and then produce characteristic disorder of motility. Kraepelin admits that the absence of true paralyses and apraxia and the slight evidences of motor aphasia in dementia præcox somewhat militate against this *near*-precentral correlation with motor disorder.

In point of fact, the speech disturbances of dementia præcox are more of a sensory than a motor aphasic character. Accordingly Kraepelin correlates them, as well as the characteristic auditory hallucinations, with the temporal lobe disorder which his literary analysis has found to be frequent. The complex speech disorders

of dementia præcox are regarded by Kraepelin as due to a weakened influence of sound-images over the expressive movements of speech or perhaps to a loosening of connection between the sound-images and concepts of objects. Auditory hallucinations are regarded as irritative phenomena of temporal lobe origin, and Kraepelin calls attention to the fact that schizophasia (*Sprachverwirrtheit*) and the tendency to the formation of neologisms are always correlated clinically with auditory hallucinations. Hallucinatory repetitions of things said and *Gedankenlautwerden* are symptoms pointing to a similar disorder in relation of ideas to expression.

As to *cortex-histology* in dementia præcox, the Munich laboratory has latterly tended to reverse the earlier conclusions as to the stratigraphy of the changes. The permanent changes, *i. e.*, nerve cell losses, are now thought by Alzheimer (as reported by Kraepelin, 1913) to be rather confined to the second and third cortical layers. The earlier work, which had been confirmed by various observers, had emphasized the gliosis of deeper layers: this is now considered to be rather a phenomenon of the acute phases of the disease.

From the new data Kraepelin somewhat hesitantly draws certain conclusions which have much in common with older contentions of the Hughlings Jackson or Wernicke type. The clinical involvement of certain higher mental functions in dementia præcox—notably that the process of *abstraction,* which serves to transform perceptions to conceptions, lower to higher feelings, impulses to higher voluntary activity—is correlated by Kraepelin with the histological involvement of the *small nerve cell layers.* Little or no use is made in this connection of the topographical idea, except that the high development in man of the upper layers in the frontal region is mentioned.

In summary of Kraepelin's present stand upon this problem, it would seem that he assumes as proven (a) topographical variations in the degree of the (microscopic) lesions, with a tendency to more marked involvement of the frontal central and temporal regions, (b) stratigraphical differences in the permanent changes found in the cortex, with a tendency to greater involvement of the outer small-cell layers. On the basis of these findings Kraepelin suggests that the major symptoms of dementia præcox may be ex-

plained (automatism, negativism, stereotypy, mannerisms, will-disorders, neologisms, speech-disorder).

Such localizing considerations represent a considerable advance (or deviation) from the classical Wundtian contentions about mind and brain. They form the great novelty of the eighth edition of Kraepelin and betoken once more the fairmindedness, readiness for innovations, and inductive power of this great clinician. The critic must observe with astonishment the logical similarity between some of Kraepelin's recent contentions and the older ones of Wernicke and Hughlings Jackson.

BLEULER'S RECENT VIEWS OF DEMENTIA PRÆCOX

Bleuler sums up his elaborate essay in its preface by saying that he has applied the ideas of Freud, of Jung, and their co-workers to the conceptions of Kraepelin. Bleuler takes dementia præcox very broadly to include the majority of those mental diseases hitherto termed functional. Yet dementia præcox is not to be taken as a mere collection of symptoms (*Zustandsbild* or symptom complex or syndrome), and the Kraepelinian conception, to which Bleuler in general subscribes, is at the farthest possible removed from the standpoint of Wernicke, for whom " to-day's motility psychosis is to-morrow's paranoia," etc. When the symptom or symptom-complex has been clearly made out, the task of the symptomatologist begins: What is the relation of these symptoms to other symptoms and to anatomical findings? What is the course of disease showing these symptoms? What are their causes? Finally, what is the fundamental disorder to which they may be reduced?

Bleuler goes the whole way of saying: dementia præcox is a disease without transitions to others. All authors tend, he says, to call the schizophrenias either an intoxication or some other kind of thing introduced *de novo* into the body. In this respect Bleuler seeks to maintain the general resemblance of schizophrenia to general paresis. Probably dementia præcox contains several subordinate diseases, much in the sense that syphilitic paralysis may be said to contain the majority of what the older alienists called dementia paralytica. There may be a few rare processes of some other nature which can evoke the same symptoms.

It is not impossible, says Bleuler, that certain slight organic disturbances in the brain may produce symptom-complexes such as we

now ascribe to dementia præcox. So also intoxication, especially with alcohol, various kinds of autointoxication and of infection, may produce the schizophrenic picture. Dementia præcox is therefore not so much a species as a genus of diseases (*cf.* organic dementia, or perhaps general paresis). We are not yet able to compare dementia præcox with, *e. g.*, infectious nephritis; we can hardly go farther than to compare it with chronic Bright's disease. Yet within the schizophrenic group Bleuler can as yet find no signs of a natural subdivision into entities: there are merely certain *Zustandsbilder* within the disease (*e. g.*, hebephrenia, catatonia, paranoid form, etc.).

As schizophrenic, Bleuler counts, besides (1) those cases commonly regarded as *dementia præcox,* also (2) cases of Kraepelin's presenile *Beeinträchtigungswahn,* (3) those cases of *manic-depressive insanity* having *truly schizophrenic* symptoms (following Kraepelin's earlier rather than his later habit in this respect), (4) *infantile schizophrenia* (though stereotypies of idiots are *not* catatonic), (5) most cases of *melancholia and mania* as these terms are used in France and England, and even the *hallucinatory manias* and *hallucinatory melancholias* of various German authors, (6) the majority of cases of *amentia* and *hallucinatory paranoia,* (7) most cases of Ziehen's *ecnoia,* (8) almost all cases of Wernicke's *motility psychoses,* (9) *primary and secondary dementias,* (10) most incurable hypochondrias, (11) some cases of Kraepelin's *Erwartungsneurose,* (12) most *hysterical insanities,* (13) certain *"nervous"* patients given to refusal of food and ideas of jealousy, (14) a large portion (but not all) of *obsessive states, Grübelsucht, impulsivity,* (15) the majority of *juvenile psychoses,* (16) insanity of *masturbation,* (17) some of Morel's *dégénérés,* (18) some of Magnan's *dégénérés,* (19) some acquired brain-weaknesses and *constitutional* insanities, (20) many cases of *moral insanity,* Kahlbaum's heboid and parethosia, Wernicke's moral autopsychosis, (21) many *prison-psychoses.*

As to *genuine paranoia,* Bleuler regards the "mechanism" of delusion-formation as probably identical with that in schizophrenia. He has had to change his diagnosis from paranoia to schizophrenia, however, in very few cases, and all these cases of altered diagnosis had shown some schizophrenic symptoms from the beginning. He leaves the question open.

As to *paranoid alcoholics*, Bleuler states that there is no evidence for the existence of an alcoholic paranoia which is not schizophrenic.

So much for the scope of Bleuler's conception of schizophrenia —a much broader one and much more inclusive than might be thought suitable at first sight. When reproached with the remark that the conception is *numerically* too inclusive, Bleuler remarks that it is a question of fact (" there are more horses than elephants, more colds than typhoid fevers! ").

As for the practical delimitation of schizophrenia from other diseases, following is a summary of Bleuler's position.

Dementia præcox, or as Bleuler states, schizophrenia, is a group of mental diseases which either run a chronic course or occur in attacks; may stop or improve at any stage, but perhaps never show restoration to absolute health. The symptoms of schizophrenia consist in an alteration of thinking and feeling and in a change of relations to the outer world. These changes are regarded by Bleuler as specific or almost specific.

In every case of schizophrenia there is a more or less distinct splitting of the mental functions. The personality of the pronounced case exhibits a loss of unity; sometimes one mental complex, sometimes another represents the personality. There is lacking all mutual correlation between the different complexes and purposes of the patient. The mental complexes do not flow together into the normal conglomerate, the impulses having unitary results, but now one complex dominates the personality and now other split-off groups of ideas and impulses dominates. Ideas are often only partially thought out, and fragments of ideas are erroneously put together to form a new idea. Concepts are therefore incomplete, lacking one or more essential components. Sometimes the ideas remain partial.

The process of *association* is determined by fragments of ideas and concepts. The resulting ideas are not merely incorrect but bizarre and unexpected. Association may stop in the midst of a thought or in progress of going over to another thought (thought blocking, *Sperrung*). Instead of a continuation of normal associative activity, new ideas come in which are incoherent with the previous train of thought.

The schizophrenic patient exhibits no *primary* disorder of perception, of orientation, of memory. Severe cases may show no emotional reactions whatever. Milder cases show that the strength of reaction to emotion is without proper relation to what the patient is experiencing. One ideational complex is responded to with intensity, another not at all.

The emotions may be qualitatively abnormal and without relation or proportion to intellectual disorder. Institutional cases are apt to show other symptoms, especially hallucinations and delusions, confusional states, dazed states, maniacal and melancholic variations in emotion, catatonic symptoms. All these are regarded by Bleuler as accessory symptoms and symptom complexes, though many of them show a specific schizophrenic character so that they aid in the diagnosis. Outside of institutions there are many schizophrenic cases in which these accessory syndromes are absent.

Bleuler describes as *sub-forms of dementia præcox*—

Paranoid.—Constantly in the foreground are hallucinations or delusional ideas or both.

Catatonic.—Constantly or for longer periods catatonic symptoms remain in the foreground.

Hebephrenic.—Accessory symptoms occur but without dominating the picture.

Simple schizophrenic.—Throughout the course of the disease only the specific fundamental symptoms are to be shown.

The fundamental symptoms of schizophrenia are a specific (schizophrenic) disorder of associations and of the emotional life (affectivity), a tendency to replace and to shut out with the patient's own imaginative experiences. Such replacement and exclusion of reality Bleuler terms autism (autismus).

Characteristic also is of course the absence of symptoms found in certain other diseases, viz., primary disorders of perception, of orientation, of memory and the like.

The ORGANIC psychoses show on the intellectual and emotional sides certain symptoms which do not belong to dementia præcox.

On the *intellectual* side dulness and slowness of perception; inability to think out complicated things completely; disorder of memory more marked for recent than for older events; disorientation for time, place and environment; disorder of attention, especially inability to pay extremely close attention.

On the *emotional* side. All the emotions are preserved to correspond qualitatively with the mental conditions. The emotions are superficial and fugitive, but not capable of giving permanent direction to the impulses.

Epileptic conditions are distinguished from dementia præcox by symptoms upon the intellectual, emotional and motor sides.

On the *intellectual side.* Epileptic conditions show either no alteration of ideational capacity or a slowing and dulling thereof. Gradual limitation of the associations as in organic psychoses, but more distinctly egocentric; slowness and hesitation of thinking processes; persistence of thinking along the line begun; circumstantiality in speaking; tendency to a certain sort of perseveration. Later there occurs a disorder of memory, much more deficient than in the organic disease. The amnesia depends apparently on physical factors.

On the *emotional* side. All the emotions are correlated with the content of thoughts. The emotional state is persistent and not plastic. At a given time the emotions of the epileptic are unitary and exhibit no lack of correlation.

On the *motor* side. Singing, hesitating speech.

Schizophrenia and idiocy.—Idiocy begins in utero or early in life, is not progressive, shows emotional variety but along normal lines (no *Affekteinklemmung*), associations limited to the "intellectually adjacent."

Schizophrenia and paranoia.—The only "dementia" sign in paranoia is the attitude of the patient to his own delusions.

Schizophrenia and manic-depressive insanity.—The latter is characterized by a uniform elevation or depression of psychic tonus affecting all three major departments of the mind (*e. g.,* feelings: euphoria *versus* depression; intellect: flight of ideas *versus* inhibition of thinking; will: excessively busy attitude *versus* general motor inhibition). There is no true dementia in manic-depressive insanity, though it may be imitated by "emotional incontinence," depressive inhibition of thought, or by dementia due to intercurrence of brain atrophy. The existence of some of these factors in a case does not militate against the diagnosis schizophrenia; the existence of schizophrenia symptoms does militate against the diagnosis manic-depressive insanity.

Schizophrenia and hysteria.—Both are characterized by their explicability on psychogenic lines and by the dominance of emotions connected with certain ideas. Hysterical symptoms occur in schizophrenic and and do not militate against the diagnosis.

Of manic-depressive insanity, of hysteria, and perhaps of paranoia, we may say (according to Bleuler) that they exhibit only symptoms that may *also* be found in schizophrenia. All other mental diseases have specific symptoms which do not occur in schizophrenia.

Concerning the anatomy of dementia præcox, Meyer says in his paper on The Nature and Conception of Dementia Præcox, "The various lesions found in dementia præcox are not clearly understood and reduced to a definite intelligible mechanism, except they are essentially degenerative or simple reactive processes" (p. 8). Again, he disagrees with Alzheimer's conception of dementia præcox as an essentially organic disease and proposes rather to term it "an incidentally organic disease" (p. 9). Again, "the histological data are not unequivocal, but mainly of a character which might as well be merely *incidental* to the functional disorders" (p. 13). Again, "the condition undoubtedly goes in some cases with a decided breakdown of cerebral material, marking an acute delirium, or perhaps an acute stupor suggesting submental factors" (p. 14). But, "the available somatic facts in most cases are by far in favor of an endogenous break of compensation of anabolism and metabolism rather than in favor of a distinct exogenous disorder" (p. 15). As to the exact meaning of *incidental* in the above usage, Meyer suggests (p. 16) that work like that of Hodge and Crile on fatgue overstimulation, and shock, may show that the nerve-cell pictures are results of functional changes.

III. Material, With Statistical Analysis

Following are statistical tables in which certain general features of the new material are presented, following the plan adopted in my paper on dementia præcox of 1910:

TABLE I.

Classified by decades according to age at death, the material shows:

2	cases	dying	in	the	second	decade	(11-20)....	1	male, 1	female.
2	"	"	"	"	third	"	(21-30)....	0	" 2	"
5	"	"	"	"	fourth	"	(31-40)....	1	" 4	"
4	"	"	"	"	fifth	"	(41-50)....	1	" 3	"
7	"	"	"	"	sixth	"	(51-60)....	3	" 4	"
4	"	"	"	"	seventh	"	(61-70)....	1	" 3	"
1	"	"	"	"	eighth	"	(71-80)....	0	" 1	"
25							(11-80)....	7	" 18	"

TABLE II.

Classified by decades according to age at onset, the material shows:

1	case	with	congenital	features	(possibly	imbecile).....	1	male, 0	female.	
5	"	"	onset	in	second	decade	(11-20).....	1	" 4	"
9	"	"	"	"	third	"	(21-30).....	3	" 6	"
5	"	"	"	"	fourth	"	(31-40).....	1	" 4	"
1	"	"	"	"	fifth	"	(41-50).....	1	" 0	"
1	"	"	"	"	sixth	"	(51-60).....	0	" 1	"
22								7	" 15	"

3 cases age at onset unknown.

TABLE III.

Classified by duration of symptoms in hemi-decade periods, the material shows:

4	cases	under 5 years in duration.....						0	male, 4	female.
4	"	between 6 and 10 years in duration.....						1	" 3	"
2	"	"	11	"	15	"	"	"	1	" 1	"
1	"	"	16	"	20	"	"	"	1	" 0	"
3	"	"	21	"	25	"	"	"	1	" 2	"
4	"	"	26	"	30	"	"	"	0	" 4	"
1	"	"	31	"	35	"	"	"	1	" 0	"
2	"	"	36	"	40	"	"	"	2	" 0	"
1	"	"	41	"	45	"	"	"	0	" 1	"
22									7	" 15	"

3 cases age at onset unknown.

TABLE IV.

SUPERFICIAL ANALYSIS OF CASES, KRAEPELIN, 1899.

Hebephrenic	4 cases,	1 male,	3 females.
Catatonic	10 "	3 "	7 "
Paranoid	11 "	3 "	8 "
	25	7	18

TABLE V.

BRAIN WEIGHTS BY DECADES IN WHICH DEATH OCCURRED.

Male....11-20,	1 case.....	1435	dementia præcox,	1376	normal (Boyd).
Female..11-20,	1 "	1270	" "	1276	" "
Female..21-30,	2 "	1148	" "	1239	" "
Male....31-40,	1 "	985	" "	1366	" "
Female..31-40,	4 "	1221	" "	1222	" "
Male....41-50,	1 "	1220	" "	1348	" "
Female..41-50,	3 "	1328	" "	1214	" "
Male....51-60,	3 "	1345	" "	1345	" "
Female..51-60,	4 "	1172	" "	1225	" "
Male....61-70,	1 "	1550	" "	1315	" "
Female..61-70,	3 "	1150	" "	1210	" "
Female..71-80,	1 "	1260	" "	1170	" "

TABLE VI.

CASES WITH DURATION UNDER 10 YEARS.

Male....1 case	1435	1357 normal	(Vierordt).
Female..7 "	1197	1235 "	"

CASES WITH DURATION 11-20 YEARS.

Male....2 cases	1238	1357 normal	(Vierordt).
Female..1 "	1145	1235 "	"

CASES WITH DURATION OVER 20 YEARS.

Male....4 cases	1329	1357 normal	(Vierordt).
Female..7 "	1241	1235 "	"

TABLE VII.

HEART WEIGHTS.

Male.....6 cases	287		
Minus 1 hypertrophy (575).....		313 normal	(Vierordt).
Female..16 cases	224	310 "	"

TABLE VIII.

Liver Weights.

Male.....7 cases	1536	1579 normal	(Vierordt).
Female..18 "	1070	1526 "	"

TABLE IX.

Spleen Weights.

Male.....7 cases	152		
Minus 1 case wt. 415	107	149 normal	(Vierordt).
Female..18 cases	101	180 "	"

TABLE X.

Kidney Weights.

Male.....7 cases	243	277 normal	(Vierordt).
Female..18 "	219	264 "	"

IV. Clinical and Anatomical Analysis of Twenty-five Cases of Dementia Præcox, being a Random Selection.

At this point I shall present (a) the condensed *clinical history,* (b) a summary of the *autopsy findings* in the trunk and limbs, (c) a transcription of the *brain findings* both on the autopsy table and by means of subsequent review in the light of systematic photography, and (d) a provisional classification as to *congenital and acquired features* in the light of all available facts, in a series of 25 cases. This series contains three cases previously studied less systematically, and presented in 1910 as Cases XII, XIII, and XIV. Another case (1297) was mentioned in 1910, but was excluded on account of a cyst of softening (the study in 1910 deliberately excluded many cases on account of complicating features); but, as the brain of 1297 was available for systematic photographic study, no reason could suffice for its exclusion from the present series, and it is included as Case I.

The cases are presented in the chronological order of their appearance in the post mortem room, as this order seemed less likely to prejudice interpretation than any other.

Case I.—F. L. (D. S. H. 6556, Path. 1297), female, of Nova Scotian stock, was regarded on admission at 29 years as a case of "*acute melancholia,*" and in fact had had a previous attack of *depression* (with

hallucinations of hearing) at 20, which lasted 18 months and terminated in complete recovery. Marriage: three children, last at 27 years. Patient went home after six months and returned again a year later in a condition termed *chronic delusional insanity* (1894). There were considerable variations in the picture in the subsequent hospital course until death at 56 years. It does not appear that the diagnosis *manic-depressive insanity* was ever considered, despite the cyclothymic tendencies of the course.

The picture was one of so-called *dementia* varying in apparent depth from year to year, and difficult to evaluate by reason of the *deafness,* which, marked on admission, grew worse. It seems pretty well established that patient had *auditory hallucinations* of a reviling nature, and she gave evidence of reaction to these for many years by excited *outbursts of profane and at times obscene speech.* But patient *always denied hallucinosis,* except at first, when she admitted *voices* but said they *did not seem real.* At first, too, she complained of a *beating and a roaring noise in her head. Delusions* were *directed both at husband and at others,* especially *mother-in-law.* Not infrequently quarreled with other patients. A good worker in the sewing-room. Wrote numerous incoherent letters.

There was *"nervousness" in her mother,* who was also said to have been *given to talking to herself.* A half-brother was said to be insane. An aunt insane.

Physically, there was early a tendency to gain weight, despite poor appetite; but later patient remained thin and grew thinner, finally dying in emaciation doubtless increased by cancer of the uterus. The *tongue* was always *tremulous. Nasal catarrh.* Chronic changes in both drums were found at autopsy.

It seems clear that this case could be described symptomatically as *paranoic* and as *allopsychic* in Wernicke's nomenclature. There seem to be *no* evidences of *catatonia,* or perhaps *any true dementia.* Where can the case be placed in Kraepelin's classification (1904), save in the *paranoid group of dementia præcox?*

Summary of autopsy findings.—Cause of death: *Carcinoma of cervix and body of uterus, with infiltration of retroperitoneal tissues.* Aortic, aortic valvular sclerosis; mitral incompetency; edema of feet and of lungs; chronic passive congestion of liver; hydropericardium; enlarged mesenteric lymph nodes; large white kidneys; apical pleuritis; ovarian and mammary atrophy; ear-drums opaque.

The findings in the head were: Hair short and scant; brown, mixed with gray. Scalp very pliable, taking but little force to remove it. Calvarium of normal thickness and contains no diploe. Dura not adherent to skull-cap even by longitudinal fissure. Dura thin, the convolutions easily discernible through it. Light adhesions between it and the pia. The *pia* is hazy along vessels. The hemispheres are equal in size; but the sulci in the motor regions, on the left side particularly, gape to the extent of 1 cm., in frontals 0.5 cm., and gaping occurs on the left orbital surface of the frontal lobe. There is an area of softening on the right temporal second convolution, 2 x 3 cm. in diameter. The right temporal lobe is

sticky to the touch. The convolutions are very superficial and thin. A recent thin white fibrinous exudate encircles the pons. The cerebellum is very much softened; weight 180 gm. Basal vessels not notable. Brain weight 1390 gm. Brain hardened *in toto* in normal formalin. Middle ears opaque. Lateral sinuses contain much blood. Cord normal. Summarized: Calvarium dense and dura slightly adherent; slight sulcal chronic leptomeningitis; left-sided cerebral atrophy; recent focal lesion of right second temporal convolution.

The *unequal atrophy* of the two cerebral cortices was most striking on the autopsy table, and the systematic review by photographs only confirms the impression. Although perhaps no area is quite free from gross atrophy when viewed from some aspect, it would appear that the entire *left* supra-Sylvian region, involving more especially the *frontal, central, and parietal areas,* is most extensively affected by the atrophy, although (if one views the right hemisphere from above) the posterior part of the *right first frontal sulcus* gapes widely and deeply, suggesting that the maximal atrophy may lie here. A study of the two mesial aspects, however, again leaves us with the impression that the left hemisphere is a little more atrophic than the right.

The superior aspect intimates that the *post-Rolandic* tissues on the *right* side are *more atrophic* than on the left. The coronal sections confirm this intimation, more particularly for the convolutions near the parieto-occipital fissure.

It is noteworthy that, despite the well-marked cortical atrophies, there was little sign of diminution in the section-area of the corpus callosum. In particular there was very slight presplenial thinning.

Also, despite the long-standing and slowly increasing deafness, the temporal convolutions failed to show convincing lesions, not, at any rate, such as to surpass those of many other areas. Nor, I assume, can the cyst of the right second temporal convolution be invoked to account for the deafness.

A woman, depressed and hallucinated at 20, depressed at 29, deluded and variously " demented " from 30 to death at 56. Outbursts of profane and obscene speech and of quarrelsomeness. Delusions allopsychic (against family). Tremor of tongue. Noises in head, deafness (peripheral).

The brain appearances seem to place this long-standing case of paranoid dementia præcox pretty definitely outside any possible temporal-lobe group of paranoic conditions. In any case, the hallucinosis was early and fleeting, and may perhaps have been related with the early peripheral deafness. As to paranoic correlations, we find supra-Sylvian atrophy, more marked on the left side. The allopsychic delusion-formation could then be correlated with either frontal or parietal disorder. It would perhaps

be pressing correlations overmuch, should we try to correlate the outbursts of (catatonic?) excitement with something in the parietal lobe, leaving the paranoia as frontal. The profane and obscene speech and the incoherent letters are consistent with the left-sided atrophy.

However, the chief value of the case lodges in the demonstration of a *non-temporal* example of *paranoia,* paranoia in which doubtless hallucinosis was not the essential agent but which consisted in a false attitude to society. On general grounds (though not on the data of this case) one might suppose this false attitude rather a frontal-lobe affair than due to disorder more posteriorly.

Case II.—From my previous study (1910) the following history is reproduced:

"XII. Woman, mill-worker (father alcoholic), at 23 had insomnia, followed by auditory hallucinations, fear, religiosity, vagrancy. In hospital, resistive, hallucinated, delusive, mute, refusing to eat; later, violence. Finally apathy, with manneristic speech and grimaces. Death from carcinomatosis, *21 years* after onset. Occipital microgyria. Chronic leptomeningitis along sulci. Cervical spinal cord firm. (*Acquired.*)"

A revision of the brain findings in the light of the present systematic study is as follows:

The *left superior frontal region* presents what looks at first like an annectant, lying in a hollow about 0.5 cm. deep from the general contour of the lobe, but with normally thin pia mater investing it. This apparent anomaly is the more striking by reason of the massive gyrus co-ordinate therewith on the right side. Both grey and white matter appear unduly narrow in this small gyrus. Tracings of the two superior frontal regions show that the surface included between co-ordinate sulci is greater on the right than on the left side.

The frontal and temporal opercula on the left side flare more than on the right, and the posterior portion of the left superior temporal gyrus disappears in the bottom of the Sylvian fissure at the anterior end of the posterior third. Frontal sections through the brain suggest that both temporal and both hippocampal regions are less well supplied with white matter than the remainder of the brain. But the left temporal region appears least well off in this respect, and the pia mater, though not appreciably thickened, peeled with some difficulty from the region, suggesting, though nowhere definitely showing, adhesions. Comparison of the two superior temporal gyri is specially tempting.

A woman, sleepless, hallucinated, apprehensive, religious at 23; later deluded, resistive, mute, sitophobic, violent; finally apathetic, manneristic, grimacing. Course 21 years.

Unlike Case I, Case II cannot exclude the temporal lobe from a share in the production of paranoia. The brunt of the lesion was undoubtedly borne by the left superior frontal and left superior temporal convolutions, both of which suggest congenital or early maldevelopment. The maldevelopment of the frontal region was probably of the nature of an interference with the development of tissues properly laid down embryologically, suggesting *hypoplasia* rather than the *paraplasia* which the temporal lobe suggests. Auditory hallucinations can perhaps be correlated safely with some sort of (functional or structural) disorder of the temporal regions.

We might accordingly correlate the hallucinosis with temporal-lobe disorder, the paranoia with frontal-lobe disorder, and the late catatoniform symptoms with a later involvement (microscopic?) of post-Rolandic tissues (occipital-lobe anomaly is suggested by the gross appearances). The case is obviously too complex for the exact correlation of symptoms with lesions; but there is in the case nothing inconsistent with the general contentions of the work in 1910 or with those of the present work.

Case III.—From my previous study (1910) the following history is reproduced:

"XIII. High-grade imbecile. Irish mill-operative. Sister epileptic. Mother of three children and a fourth illegitimate. One attempt at suicide? Eight years' course, with onset at 23. Religiosity. Could see Virgin Mary at any time (hallucination rather than delusion). Imitation of saints' attitudes as seen in pictures. Possible gustatory hallucinations. Death at 31. Unequal pupils. Superior temporal anomaly. Cervical spinal cord unusually large. Slight leptomeningitis, especially basal. (*Acquired and congenital.*)"

A revision of the brain findings in the light of the present systematic study is as follows:

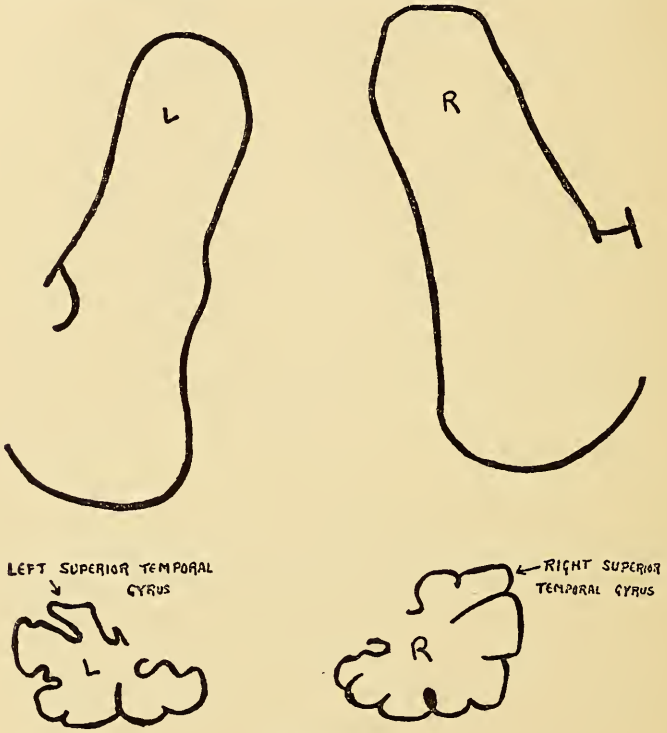
The most striking feature of this brain—and that which caught attention in the former publication, where this case appeared as Case XIII—is an anomaly of the left superior temporal gyrus, which is almost entirely hidden in the middle part of its course by the middle temporal gyrus. Tracings of the contour of the two temporal lobes, laterally viewed, suggest that there is an actual undersize to the left region as compared with the right. (Text-fig. 1, p. 608.)

Tracings made from appropriate frontal sections demonstrate the same fact. (Text-fig. 1, p. 608.)

It is clear that the amount of white matter in the temporal lobes is far less on the left side than on the right side.

The lateral ventricles are moderately dilated, but the left much more so.

A woman, regarded as possibly a high-grade imbecile, showed hyperreligiosity at 23. Voluntarily induced visions (Virgin Mary). Assumption of saints' attitudes. Religious songs sung.



TEXT-FIG. 1.

Here the correlations are not too strongly in evidence. Clinically we must remain in doubt whether the attitudinizing was catatonic or paranoic in origin. In fact, the history is not full. I get the impression, however, that there were numerous sensorial elements in the case (bad tastes, visual hallucinations). There was considerable amnesia (imbecility, however). The moderate *internal hydrocephalus,* more marked on *left* side, coupled with the *left-temporal-lobe hypoplasia,* suggests more a correlation with

certain imbecilic features than a correlation with the acquired mental disorder. We should greatly desire to find in the history of this case some auditory basis for her symptoms. On the whole, however, there is more visual basis than auditory.

CASE IV.—From my previous study (1910) the following history is reproduced:

"XIV. Shoe-operative. Attack of doubtful character at 12 years. Paranoid symptoms began at 18. Catatonic symptoms gradually supplanted the paranoid. Death at 32. Spinal cord unusually small. Brain weighed 985 gm., but was apparently not to any great degree atrophied. Heart (145 gm.) and liver (945 gm.) also small. (*Congenital.*)"

A revision of the brain findings in the light of the present systematic study is as follows:

The brain (weight 985 gm.) of this case was first described—under Case XIV of the former communication—as "not to any great degree atrophied." On account of the microcardia and microhepar, the suspicion was that the case was congenitally defective (this despite the fact that the subject is said to have been an adequate shoe-operative). More extended study calls attention to the fact that the occipital, and perhaps still more the temporal, regions are more atrophic (or aplastic?) than the rest of the brain. This difference is noticeable both in the external appearances and in the frontal sections.

A man, confused (and unconscious?) at 12; sleepless, "mesmerized," violent, suicidal, at 18; fantastic delusions, fantastic transitivism; profanity, obscene speech; later pronouncedly catatonic (special attitude).

Correlations cannot be made on account of universality of lesions. If anything, the paranoia might be regarded as post-Rolandic or sensorial in origin. Possibly the fantastic type of delusions will prove more often post-Rolandic than pre-Rolandic; for such delusions are on general grounds more nearly allied to normal exercise or overexercise of the imagination than to the assumption or establishment of an abnormal attitude to normal ideas. Thus, on general grounds we may conceive a more "impressional" type of delusions and a more "expressional" type, the former more apt to be post-Rolandic, the latter more often pre-Rolandic in origin. The patient under consideration obviously had the power of conceiving and probably of imagining more complex ideas and situations than a normal person. Suppose the post-Rolandic and infra-Sylvian emphasis of the lesions in this case to be roughly correlative with the fantastic (sensorial) delu-

sion-formation; then the later development of catatonia would be not unnatural, because kinæsthetic disorder would be apt to ensue from a spread of the process to near-by parietal regions.

The case is surely allied to cases of the new group *paraphrenia phantastica,* which Kraepelin has separated out from the other cases of dementia præcox. Kraepelin, however, remarks that about 40 per cent of his cases of dementia paranoides (as described in the seventh edition, 1904) turn out essentially cases of demenita præcox in the ordinary sense, and accordingly do *not* turn into the new (1913) group of paraphrenias (analysis of Zerko). Perhaps, then, we would best regard this case as one of paranoid dementia præcox, suggesting paraphrenia phantastica, but proving by its outcome its closer relation to dementia præcox (*i. e.,* by the catatonic features of the terminal condition).

See further discussion under Case VII.

Case V.—M. W. (D. S. H. 13704, Path. 1383) was an old Scotch woman, a pensioner (husband in Civil War), of whom all history was lacking, but whose mannerisms, ways of thinking, and amnesia suggested the diagnosis *dementia præcox.* The examiners were inclined to suspect an *alcoholic* admixture in the dementia. Her age even was doubtful, but was at least 62 at death.

Untidiness, resistivism, loquacity, repetitive talk, loud singing, perseverant "Open door, open door," spasmodic movements of lower jaw, lid-closure, violence to patients, mischievous acts, silly smiling, gluttonous eating, disorientation for time and place.

Occasional more lucid intervals with euphoria and long narrative conversation about mantel-piece, tea-canister and a clock once possessed by patient.

Death at about 62 from *bronchopneumonia.*

Summary of autopsy findings.—Emaciation; general arteriosclerosis; various chronic lesions: cystitis, nephritis, apical tuberculosis, pleuritis, pericholecystitis, perisplenitis. Chronic internal hemorrhagic pachymeningitis of *right side.*

Head: Dura markedly adherent to calvarium on right side. Brain weight 1060 gm. (60 gm. loss, according to Tigges' formula). Marked diffuse chronic leptomeningitis, most marked over right Sylvian and Rolandic regions. The moderate atrophic process, like the thickening of pia mater, is more marked in pre-Rolandic tissues and on the right side. The frontal atrophy is better marked mesially than laterally. The sulci at the junction of the right superior and middle frontal gyri are deep. Both Sylvian fissures and the right first temporal sulcus are deep. The corpus callosum is thinned in an unusual place (in the present series of examinations), viz., in the region just back of the rostrum. Moderate to marked internal hydrocephalus, especially of mid portions of ventricles.

A woman whose psychosis is classed as dementia præcox on the basis of the terminal condition. The diagnosis is accordingly dependent on accepting at its face value Kraepelin's statements about *characteristic* end-states. No suspicion as to the duration of this case can be risked. There was such a general distribution of lesions in the areas with which we are particularly concerned—frontal, postcentral (Rolandic) and temporal regions—that no correlations can be safely made. There is, however, in the findings, nothing inconsistent with our general contentions. Such long-standing cases are rarely suitable for correlations.

We have no definite evidence of hallucinosis or delusions in this case; but the case should not be counted one way or the other in topographical analysis. Its chief or only value must lie in showing the nature and spread of the mild lesions that may be shown by a terminal case of what was probably dementia præcox. The suspicion of alcoholism must be remembered in connection with the general Rolandic (precentral as well as postcentral) atrophy.

Case VI.—C. B. (D. S. H. 14779, Path. 1413) was a boy of 15 when he was committed to D. S. H., and died there a year and four months later of *typhoid fever, ulceration, and peritonitis.* The mental diagnosis was eventually considered to be *dementia præcox.*

Mother nervous and melancholy during her pregnancy with this boy; hard usage and neglect by husband complicated the pregnancy. From an accident to the boy at seven, is said to date a change of character, with refusal to go to school, fretfulness, and nervousness. At 14 another accident to back and head. An inmate of a truant school for two periods of three and two years respectively. Uncle a patient at state farm.

Auditory hallucinations, refusal to eat, nervousness and certain delusions antedated commitment by only a week. (Masturbation had begun four or five months before, but had probably ceased.) Insomnia is said to have lasted four nights after onset. *Food poisoned. Passers-by or Indians were to kill him* (given to blood-and-thunder stories). *"Had never done anything to gladden America," "Traitor," "Going to be shot."*

Cyanosis of hands. Gait staggering (no Romberg); right cremasteric reflex not active.

After eight months' observation it was thought that the patient really belonged in the moron or subnormal group of defectives, and in the quasi-criminal class. A little over 11 months after the delusional episode upon which he was committed, patient developed a spell of disturbance which lasted 10 days. In this spell, patient was noisy, threatening, profane, denudative, given to disconnected and incoherent talking, lost weight and grew pale. Almost three weeks later typhoid fever developed, and death followed from peritonitis.

Summary of autopsy findings.—Aside from the typhoid fever lesions, there were bronzing of skin; unusually small adrenals (questionable glycosuria had appeared on entrance, but later disappeared); a small thyroid; slight sclerosis of aorta and right auricular endocarditis; cardiac hypertrophy; chronic myocarditis, pleuritis and pericarditis.

The brain weighed 1435 gm. (over weight 90 gm. by Tigges' formula). There is nevertheless a tendency to *frontal atrophy,* perhaps more marked on *left* side. Striking anomalous folding of convolutions in both callosomarginal regions above the splenium; but on the left side the fissuration suggests acquired lesion rather than congenital anomaly (region above and behind the rostrum).

A boy, truant school inmate five years; hallucinosis, refusal to eat, delusional episode at 15. Possibly moron, or subnormal, or quasi-criminal. Some symptoms of pluriglandular disorder. Catatonic excitement for 10 days.

On account of the comparatively brief duration of this case (16 months), it might well be imagined that the brain would not have registered coarsely any extensive lesion. The brain was, as noted above, probably somewhat over weight. There is a tendency to bilateral (more left-sided) frontal atrophy or aplasia, which may perhaps be correlated with the conduct-disorder, and with which in some partial way the delusional episode could perhaps be connected. More interesting is the callosomarginal lesion, which so far suggests an acquired feature.

This case is to be subject to especial analysis from the moron standpoint.

Case VII.—F. C. (D. S. H. 8161, Path. 1472) was a case of so-called "primary delusional insanity" in a teacher with normal-school education who slowly developed *fears of abduction,* and already at 28 had *fear of her murder by Catholics.* At 30 was examined by alienist and described *hearing signals* and *seeing strange actions* (ideas of reference rather than hallucinations?). *Soul swept 10 feet from her body by stream of electricity. Tormented with electricity by persons under the eaves. Mother poisons the boarders. Children taken into a house to have heads compressed.*

After admission at 40, soon became somewhat euphoric and very busy. Delusions concerning special treatments which she is receiving (hypnotism, massage, moulding of head). If she got a *newspaper* from a certain patient, it was *really a million dollars for which she was selling her soul.* Stopped eating meat and said she had acquired *"the thought language, i. e., false hearing, a divine inspiration,"* also had become *able to talk with beasts, birds and insects. Parents had brought up children for scientific experiments* and given them away.

Aside from refusal of food on the basis of delusions, no actions surely interpretable as catatonic developed until 48 (peculiar stepping movements).

At 50, frequently hallucinated (*Jesus standing by*). Formerly deluded concerning *marriage to a Mr. King,* now she was *married to the Prince of Wales, later to Christ.* Carried *bundles tied to waist, as she had no little children pulling thereat.*

Purulent sinuses of breast at 54. Body weight reduced to 63 pounds.

Summary of autopsy findings.—The cause of death was *phthisis pulmonalis, with necrosis of ribs and cold abscess of breast; tuberculous enteritis, with ulceration; tuberculosis of spleen; mesenteric and general enlargement of lymph nodes; sacral decubitus; acute nephritis.*

Among chronic lesions were atrophic breasts, uterus, and ovaries; enteroptosis; pericholecystitis; perisplenitis; pleuritis; myocarditis; renal infarct.

The brain weighed 955 gm., a reduction of 220 gm. if Tigges' formula can be applied. The brain was described as "infantile" by the pathologist, Dr. A. H. Peabody, although from the clinical history it seems clear that the early education of the subject required more than an infantile brain. As against aplasia, the clinical history certainly suggests that we should be dealing with atrophy. Perhaps the *increase of consistence* noted on the autopsy table is in line with the diagnosis atrophy. There is little or no dilatation of ventricles, and the corpus callosum appears in good proportion to the remainder of the brain. The hemispheres are somewhat peculiarly shaped in that the occipital poles are much tapered off. The *gyri* bordering the *Sylvian fissures* are more *atrophic* than the rest of the gyri; next, perhaps, the *middle frontals.*

A woman with a 26-year course of paranoic symptoms beginning at 28. Hallucinosis or possibly ideas of reference of auditory content. Movements possibly catatonic at 48.

Whether this be a case allied to *paraphrenia phantastica* or a case better termed dementia paranoides (in the sense of Kraepelin, 1913), we must be interested in the fact that the case agrees in several respects with Case IV. Both cases show remarkably atrophic or aplastic brains (IV 985 gm., VII 955 gm.). Both cases have a history of complete social adequacy (IV a shoe-operative, VII a normal-school graduate teacher). Both agree in the possession of temporal-lobe atrophy (or aplasia?) more marked than that elsewhere. Both possessed delusions of a type which I provisionally termed (see Case IV, above) "impressional" or sensorial, and presumably more closely related to post-Rolandic and infra-Sylvian cortex than to pre-Rolandic supra-Sylvian tissues. There was a tendency to greater atrophy of the middle frontals than elsewhere, in Case VII.

Case VIII.—H. P. (D. S. H. 16166, Path. 1487) was a case regarded as a *catatonic dementia præcox,* although the whole course lasted about three weeks, terminated by *diphtheria.* There were no symptoms referable to the diphtheria, except some choking sensations a few hours before death at 34 years. (The diphtheria affected the tonsils and epiglottis, and the typical organism which was recovered was able to kill guinea-pigs in 24 hours.) The onset is said to have been with brooding over a death in the family.

The attack started with religiosity ("lost," "*very bad*") and talk about God and the devil. Excitement and occasional violence.

In hospital: resistivism, mutism, confusion, involuntary passing of water and feces. Looked worried, anxious, surprised. Could rarely be made to lie down. Insomnia, anorexia. At first the pulse was 120, high tension and volume (during last three days, pulse became normal). General tremor of the body, especially of the lips. Excessive growth of hair in axilla, hair over sternum. Obese.

Summary of autopsy findings.—Cause of death: *Diphtheria, bronchitis, acute pleuritis.* Also patent foramen ovale, persistent thymus, anteflexion of uterus, asymmetry of face.

The brain (weight 1300 gm.) was asymmetrical (to correspond with the face), and showed especially a somewhat acutely tapering left occipital lobe (less marked, right). The frontal regions were full and fairly complex (exceeded by six other cases, right; but by 11 other cases, left). All the sulcus counts showed the right hemisphere more complex than the left (five areas). The fore-and-aft measurements showed the pre-Rolandic region far in excess of the post-Rolandic.

Despite these features, no focal emphasis of any lesion could be made out in the gross (neither sclerosis nor atrophy); and, while from an anthropological point of view doubtless far from negative, the brain must be regarded, from the narrower pathological point of view, as *negative.*

A woman of 34, with a total course (the shortest in the series) of three weeks, terminated by diphtheria. The case was regarded as catatonic dementia præcox, though perhaps the diagnosis is forced. A brief period of delusions was succeeded by symptoms of an exhaustion character among which resistivism and mutism probably gave rise to the diagnosis catatonic.

Whatever the correct diagnosis (it is even conceivable that the case is an obscure example of symptomatic psychosis), there seems to be some anthropological evidence (asymmetry) for supposing a congenital or early acquired basis for the development of dementia præcox.

Case IX.—C. P. (D. S. H. 15546, Path. 1491) was a woman of wholly unknown antecedents, who died after a little over a year of symptoms hardly interpretable except as *catatonic.* Presumably a case of *dementia præcox.* Death at 53.

Persistent posture in bed, head held at right angles to trunk, eyes staring fixedly at space. Mutism, cerea flexibilitas, resistiveness, occasional negativism, eyelids not closing on approach of finger, untidiness. Before admission she admitted some ideas of being poisoned, but after admission was never got to say more than " I don't know. I can't tell you."

Summary of autopsy findings.—Cause of death: *Hypostatic pneumonia.* Bronchial lymphnoditis; bilateral obsolete tuberculosis and fibrous pleuritis; chronic pericholecystitis (old operation scar) and interstitial hepatitis; chronic gastritis and enteritis; mitral and aortic valvular endocarditis and chronic epicarditis; aortic sclerosis and sclerosis of coronary, internal mammary, external iliac, and basal cerebral vessels; umbilical hernia; retroversion of uterus; left ear-drum opaque.

Head findings: Dense thick calvarium except in fontanelle region. Dura and pia normal. Brain weight 1330 gm. (over weight by 50 gm. according to Tigges' formula). This brain was examined *three days post mortem.* The hippocampal gyri were firm; the occipital, frontal, and temporal regions were all softer (and in that order).

Systematic examination of the brain showed the left hemisphere longer and shallower than the right. The tip of the left temporal lobe is pointed. Section showed presplenial thinning of the corpus callosum. The mesial views suggest atrophy, particularly about the paracentral lobule. Chronic internal hydrocephalus.

A woman dead at 53, whose psychosis is classed as catatonic dementia præcox on the Kraepelinian basis of characteristic terminal state (compare Cases V, XV). An over-weight brain was coupled with a marked internal hydrocephalus.

Correlations need not be pushed, seeing that the history is obscure. In the history, however, we have some little evidence of delusions as well as marked catatonia. With the latter we may possibly correlate the mild atrophy (aplasia?) of the paracentral lobules.

The case will be further considered under the heading hydrocephalus.

Case X.—M. P. (D. S. H. 15083, Path. 1507) was a case of somewhat doubtful diagnosis regarded as *paranoid dementia præcox.* There was an *hereditary taint* (paternal uncle a suicide at 36, an aunt insane for 30 years, a brother a suicide at 21). Menstruation at 20. Onset of mental disease resulted from a love affair. Inmate of two hospitals in New York State, from 24 to 28. Afterward unimproved, melancholic. Admitted to D. S. H. at 58, apathetic, deluded concerning brother and his wife. Masturbation observed at times. Mannerisms of speech.

Death at 60 from *generalized carcinosis,* a year and two months after removal of breast and pectoralis. Metastasis in brain.

Summary of autopsy findings.—Emaciation; scar, thorax; rigor mortis; edema, legs; arcus senilis; adhesions, appendix and spleen, colon, liver, lung; arteriosclerosis internal mammary, bicuspid and mitral valves, aorta and internal iliacs; bronchopneumonia; carcinoma of lungs, liver, brain; interstitial nephritis.

Head findings: Calvarium thin and soft. Edema of brain. Brain weight 1280 gm. Pons and cerebellum weight 145 gm.

Small depressed areas in postcentral gyrus bordering on median longitudinal fissure. Inferior temporal convolution on each side somewhat softer than usual. In one of the blood-vessels in the postcentral sulcus, right side, is a small amount of granular material. Brain throughout very edematous. Sulci show a slight tendency to gaping, especially in the temporal region. Floor of fourth ventricle smooth. Middle ears negative. Cord shows no gross adnormalities except a marked constriction in mid-dorsal region, which may have been caused in removing.

The third right frontal gyrus, anterior to temporal tip, contains a tumor 1 cm. in diameter, in the grey matter, and easily enucleated. In the precentral gyrus (right), superior portion, are two—the smaller 0.5 cm. in diameter, extending to white matter; the other 1.5 cm. in diameter, on the surface and extending 2 cm. into the cortex. In the precentral (right), lowest portion, is another, 1.5 cm. in diameter, not visible on surface but extending into the grey matter. Hippocampal gyrus (left), 1 cm. in diameter, tip. Occipital lobe (left), 1 cm. in diameter, lobus cuneus.

A woman who began to menstruate at 20, and who appears to have been insane from 24 to death at 60. Masturbation observed after age of 58.

Correlations are difficult by reason of the intercurrent brain metastases. Aside from these, the temporal regions appear to be anomalous and unduly simple in architecture. Only one other case (1574) has as simple a construction. This is the more remarkable, because the other regions whose sulci were counted are well up to the normal in complexity. The observation at autopsy of gaping sulci in the temporal regions emphasizes the simplicity of convolutional pattern here mentioned. The suggestion is that the region was laid down in the embryo with undue simplicity and later became subject to a moderate differential atrophy. No evidence concerning hallucinosis in this case has been as yet obtained. The frontal regions suggest an atrophy or aplasia of milder degree.

Case XI.—G. P. (D. S. H. 10378, Path. 1509) was a case of *catatonic dementia præcox,* with onset at 21 of "acute melancholia." Data available concerning years from 48 to 58. The "attack" at 48 was said to be the sixth.

Hyperreligiosity, exaltation or indifference, excitement and impulsive acts, mutism, resistivism, negativism, refusal of food, mannerisms, characterized the last 10 years. Once secreted knives from dining-room on his person. Auditory hallucinations were suggested by some of patient's attitudes. No cerea flexibilitas, but for days at a time limbs were absolutely limp, opposing neither active nor passive resistance to motion.

Autopsy 24 hours post mortem.

Summary of autopsy findings.—Cause of death: *Tuberculous enteritis and miliary tuberculosis of lungs (also pleuritis) and kidneys.*

Acute or active lesions: Acute cystitis; sacral and left gluteal decubitus; abrasions and ecchymoses of right eye; edema of brain.

Chronic lesions: Emaciation; lymph-node enlargement, mesenteric, bronchial, inguinal, epitrochlear; peritoneal and pericardial effusion; aortic, coronary, internal mammary, pelvic arteriosclerosis; chronic pleuritis, pericarditis, perisplenitis, pachymeningitis; anæmic bone marrow of femur; fatty and cirrhotic liver; chronic mitral and tricuspid endocarditis.

Anomalies: Left middle finger amputated; asymmetry of face (left eye and ear elevated); patent foramen ovale; fetal lobulations of spleen; constriction of stomach.

Nervous system: The brain, by Tigges' formula, should weigh 1365-1370 gm., but actually weighs 1265 gm.

This brain shows a moderately well-developed corpus callosum with little or no trace of presplenial thinning. However, there is a distinct though moderate *dilatation of the lateral ventricles.* This dilatation probably corresponds with a moderate general atrophy of the *frontal, central, and parietal,* as well as the greater part of the *mesial convolutions,* about equal on the two sides. The anterior part of each superior temporal gyrus is somewhat withdrawn from the surface of the adjacent gyri.

A man dying at 58 after a course of 37 years. Correlations cannot be safely made on acount of generalized lesions (see also below under hydrocephalus). Paranoia, catatonia, hallucinosis, were all clinically in evidence; ample and suitable lesions exist for correlation.

Case XII.—M. C. (D. S. H. 8469, Path. 1518) was a woman stated to be hereditarily insane. "Dementia" showed itself at 16. Hospital treatment 20 to 22 (suicidal). Again under medical care (depression) at 26 and admitted to D. S. H. Emotional, capricious, abrupt in talk, once attempted suicide (masturbation). Boarded out two months until again restless and without appetite. On return, improvement. Periods of a few days of persistent masturbation. Periods of excitement. For some years would at times tear paper up in little bits and scatter it about. At 41 had become apathetic and careless about person. High-pitched, unmodulated, whining voice. Asked how she was, replied "I don't want all my teeth pulled." Asked how she was getting along: "It's fair weather." At 43 negativistic, resistive, laughing to herself, answers (except a very few) irrelevant,

destructive to clothing, scolding. At 44, acquired a habit of going to wash-room to wet hair, dress, and skin, refusing to go to bed. Phthisis had begun at 42. Death at 45.

Summary of autopsy findings.—Cause of death: *Ulcerative gastritis and enteritis.*

Acute lesions: Subpericardial hemorrhages.

Chronic Lesions: Emaciation; tuberculosis of left lung; chronic nephritis; aortic and internal mammary arteriosclerosis; mitral endocarditis; cholelithiasis; fatty liver; healed decubitus; fibromyoma of uterus.

Nervous system: Brain weight 1385 gm. (over weight by Tigges' formula, 8 x body length, 160 cm. = 1280). No lesions or anomalies were observed at the autopsy table, but on median section certain features were discovered. The most striking features of the brain are:

(1) A marked *presplenial thinning of the callosum* (which in general, however, is well-developed).

(2) An unusual appearance of *puckering and wrinkling of the right callosomarginal gyrus* in the long horizontal portion superior to the main part of the callosum (the latter condition resembles in a lesser degree the condition in 1413). The corresponding region of the *left* callosomarginal gyrus in 1518 shows several small sulci not found on the right; it is accordingly hard to avoid the hypothesis that the condition of the right depends on some developmental, congenital, or early factor. Its superficial resemblance to the gross appearances of certain other convolutions in certain cases supposed to have been victims of *early encephalitis* suggests a similar hypothesis for this case.

(3) The right hippocampal region both is superficially less extensive than the left and also, on cross-section, exhibits a retraction from its proper plane, due largely (it would appear) to the smaller size of the right cornu ammonis but perhaps also associated with a thinning out of the hippocampal white matter.

(4) There is a definite though slight degree of *chronic internal hydrocephalus,* more marked in the posterior cornua of the lateral ventricles. There is, however, no appreciable difference in the degree of dilatation on the two sides—though this might perhaps have been predicted if the causes of the hydrocephalus acted simultaneously with those of the hippocampal irregularities mentioned under (3).

(5) Very striking, in relation to the *right-sided hippocampal aplasia,* is a *left-sided frontal aplasia.* Counts of the sulci show that the *left frontal region* is *simpler* than the right (16: 19), despite the fact that it is *smaller in volume* than the right. The other regions counted show no marked differences.

This case is to be subjected to more special study.

A woman with 29 years of symptoms, at first coming in attacks, later permanent. It is a question where to classify the case; on the whole, it would appear that the case became in the end catatonic. However, there were numerous evidences of paranoia.

I will not attempt to make correlations in this case, by reason of forthcoming more extensive studies. The peculiar schizophrenic irrelevance of replies should perhaps demand a temporal-lobe correlation not specially indicated in the above description; but the hippocampal disturbance may possibly involve the temporal region in ways which future study will reveal.

Case XIII.—L. W. (D. S. H. 12692, Path. 1540) was a Newfoundland table-girl of Irish extraction, Catholic, who began to grow careless and to show poor judgment in little ways at about 20 years, but first showed pronounced mental symptoms some two months before admission at 23. Five years later epithelioma of the tongue developed and death followed in some nine months at the age of 29. It is not clear that this epithelioma had at all to do with the mental symptoms, despite the appearance of a small metastasis in the pituitary body (no glycosuria at any time, although no tests later).

The onset was with headaches, erratic conduct, apparent apathy, and delusions possibly of a sexual nature touching a mere acquaintance. Patient preserved an evasive and reticent attitude regarded as paranoidal until death, and remained as a rule quiet and apathetic, with occasional periods of irritability and restlessness. Careless as to dress, she was as a rule tidy in personal habits.

Impulsive striking at patients or attendants was attended with facial flush. Profane, not obscene.

Auditory hallucinations were surmised from certain reactions. Occasional talking to herself, silly, smiling, lying prone on couch, or standing for long periods against the wall. Was occasionally got to work on the ward or in the laundry for a few days.

It is interesting that, in addition to a fine tremor of extended tongue and fine tremor in handwriting, Dr. F. Robertson Sims found exceedingly lively knee-jerks and a bilateral ankle-clonus, more marked on the right side, upon the initial examination.

Memory defects were largely of an extrinsic order, being due rather to inattention and apathy than to demonstrable deterioration. On the other hand, there seems to have been a true deterioration in calculating ability (patient formerly able to make change properly); but how to interpret this is doubtful.

Inattention, apathy, allopsychic (probably autopsychic) delusions, defects of judgment, irritability, impulsive violence, resistiveness, seclusiveness, and postural mannerisms formed a picture possibly suggestive of catatonic dementia præcox, although very probably the onset of the case should be taken to indicate a paranoic condition with some sexual nucleus.

The Wassermann reaction in serum and fluid was negative, and the fluid was cytologically negative.

Summary of autopsy findings.—Death was due to *tuberculous pyopneumothorax,* besides which there were epithelioma of tongue, with micro-

scopic pituitary metastasis, and submaxillary metastasis; emaciation and scaphoid abdomen; chronic endo- and myocarditis; chronic peritonitis (liver and spleen); coronary arteriosclerosis, slight; chronic and acute nephritis; mesenteric lymphnoditis.

The brain weighed 1165 gm. According to Tigges' formula, the brain should have weighed 1240 gm. (subject 155 cm. long). *Pial thickening over vertex.* Brain firm. The *superior frontal regions* of both sides show a mild degree of *atrophy* (or aplasia?). The mesial surfaces of the prefrontal areas show somewhat deep hollowing of sulci. Perhaps the *left angular gyrus* region may be thought to show the greatest degree of *atrophy* (or aplasia?). The corpus callosum is of proper dimensions and without thinning.

The whole brain gives the impression of a mild atrophic (and proportionately sclerotic) process which has affected the areas above mentioned only a little more than the remainder.

The right temporal area is the simplest in construction found anywhere in the present series, if the sulcus count can be trusted. As in Case XII, there is a degree of internal hydrocephalus, and here still more sharply to the posterior cornua of the lateral ventricles.

A woman with largely paranoic symptoms for six or seven years, and with mental change beginning insidiously at 20. Death at 29. The question between paranoia and catatonia is reflected in the autopsy findings. Both anterior and posterior association areas are affected (superior frontal and angular gyrus). Correlations can hardly be safe, on account of the generalized character of the lesions.

Case XIV.—N. H. (D. S. H. 16016, Path. 1571) was a case regarded as *paranoid dementia præcox.* Thought to be "going to pieces" at 45 to 46. A "rolling stone," sometimes in fairly good circumstances. Academy student three years (failed to graduate), reporter, book-agent, insurance agent, theatrical work, bicycle business, Spanish-War veteran. Syphilis at 28. Married at 41, later divorced.

Somatic delusions: *scalp tied down, hair growing in. "Hair backed up behind my neck as though it was caught behind my ears. Kept growing and growing until it sort of solidified, making one side more noticeable than the other."* (Thought he resembled Sir Henry Irving, especially with regard to hair.) *Rectum out of line. Whole body drawn toward the left,* so much that patient could *"feel a great flap under left arm."* Picked at sores (some, results of self-mutilation).

After six months, a certain improvement; ideas about body corrected, except that hair still made him feel uncomfortable, and he still picked at sores on body. Later felt a *tightness of chest.* Death at 54.

Summary of autopsy findings.—Cause of death: *Bronchopneumonia.*

Acute lesions: Conjunctivitis, otitis media, abrasions.

Chronic lesions: Aortic and coronary arteriosclerosis, mitral and aortic

valvular endocarditis, cirrhosis of liver (1000 gm.; stellate scars), constriction of œsophagus, scars of apices of lungs, subcapsular cysts of kidneys.

Nervous system: The scalp is not unusual. The calvarium is asymmetrical, measuring 3 mm. in the frontal, 3 mm. in the temporal, and 5 mm. in the occipital region. There is a very small amount of diploe. The tables are thin. There is some asymmetry in the floor of the skull. The left frontal fossa seems flatter than the right, the right being more pointed anteriorly and sloping back more acutely than the left. The crista galli is deviated to the right from forward to backward. The left temporal fossa is broader than the right; the bone bulges out more on the left side than on the right. The right occipital fossa appears larger than the left. There is no particular deviation of the sinuses. The lateral sinuses are clear. The right middle ear shows considerable injection, and there is a small amount of serous, possibly purulent, material there. The brain weighs 1490 gm. (over weight by Tigges' formula—8 x 165 cm. = 1320). The pia is slightly clouded over the sulci and over the vertex. The pial vessels are somewhat injected. There is considerable Pacchionian granulation. The brain is fairly symmetrical, and everywhere firm. The convolutions are well rounded and there is no gaping of the sulci. There is slight flattening of the convolutions in the precentral region on the right side, more than on the left. The temporal lobes are the softest portion.

The pons and cerebellum weigh 175 gm. The ependyma of the fourth ventricle is smooth. The pons and cerebellum are firm.

Paranoid dementia præcox. Delusions about scalp and hair, bodily distortion, possibly due to sensory impulses from a marked cranial distortion or to local cerebral pressure (left temporal lobe accommodated in less space than right, flattening of right precentral region—somatic delusion of *great flap under left arm.*) There were various effects of syphilis (of which there was a history at 28, 26 years before death), such as stellate scars of liver and œsophageal constriction (somatic delusions of *tightness in chest*).

The interpretation of this case would be easier if we could suppose that the skull distortion and brain pressure were responsible for some of the symptoms (compare my paper, " The Somatic Sources of Somatic Delusions ").

The sulcal counts of this case indicate the comparative general simplicity of the gyri.

Case XV.—A. C. (D. S. H. 16669, Path. 1574) was a case observed in hospital less than three months, and in which the diagnosis *dementia præcox* was only a retroactive one. The patient, a well-educated single woman of American stock, was the eldest of five children, all regarded by some as peculiar (one brother unbalanced and unable to take care of himself); and her father was a suicide at 43 in a despondent fit (mother died of cancer).

Patient always an invalid. Scoliosis. Left talipes equinus, shortening and atrophy of left leg, arthritis of left knee with condylar hypertrophy, left knee-jerk sluggish. Flattening of left occipital region of skull was a striking feature during life. Somewhat deaf (purulent otitis media, right, at autopsy).

Melancholy spells off and on *from the 40th year* (poison purchased at times). After 55, conceived exaggerated *ideas of wealth* (gave pipe organs to churches, and handed 20-dollar bills out when her fortune did not permit this). At 59, there was an *appearance of dementia* (unkemptness, untidiness, lack of spontaneous conversation, monologue about fairies) and possibly some actual losses of school knowledge. Restlessness, noisiness, and loquacity preceded her admission to hospital.

Impairment of vision (no arcus senilis). Aside from slight *tremor of tongue,* no neurological disturbance.

Autopsychic delusions: *"Not in human form," "I try to make myself into a fairy, but I can't vanish myself,"* "Feels strange." Sometimes thought people about her were *scarlet fairies.* Said she had a *choking feeling in neck* (cf. *cystic thyroiditis* found at autopsy).

Summary of autopsy findings.—The cause of death is doubtful, but the atrophic spleen (16 gm.) and the *atrophic intestinal mucosa* and *enlarged mesenteric lymph-nodes* suggest the terminal process. The brain tissue was a little soft. Acute cell changes in three levels of spinal cord, and diffuse Marchi blackenings. There were also a mild *pneumonic* process in the right lung; emaciation; gastroptosis; chronic aortic endocarditis; slight sclerosis of aorta, common and internal iliac arteries, and basal cerebral arteries; chronic parenchymatous nephritis.

The brain (1235 gm., over weight by about 90 gm. according to Tigges' formula; but the scoliosis had reduced the body length) was of a normal appearance in the gross, save for pial thickening over the vertex. There was a slight *presplenial thinning of the corpus callosum.* The *frontal and parietal opercula* of the left side show a slight retraction indicating a possible slight atrophy. The superior temporal regions are well developed and possess more cross-subdivisions than usual.

The cephalic asymmetry is reflected in the brain in the form of a slight flattening of the left hemisphere as if by a force operating inwards and downwards from the left.

It is not clear that this case can safely be termed one of dementia præcox on the score either of its course or of its terminal phenomena. It may more properly belong in one of the more obscure *presenile* groups, in which a hereditarily tainted person may be predestined to fall victim to a process of unknown character, possibly resembling that of *manic-depressive* insanity.

Case XV was another case (compare Cases V, IX) of diagnosis "dementia præcox" retroactively made. There is no evidence

of either hallucinosis or catatonia in the case. It is not certain that the case is one of dementia præcox (see above). The delusions of grandeur taking effect in overt extravagant acts and the fantastic autopsychic delusions can be related possibly to some process barely indicated in the gross (frontal and parietal opercular atrophy); it is striking that the temporal lobes look perfectly normal.

Case XVI.—A. D. (D. S. H. 14453, Path. 1575) was an *unclassified* case, thought to be of the *dementia præcox* group. The patient was of an old New England family and comparatively well-educated (Chautauqua supplement). Hotel housekeeper, matron of orphan asylum, later dressmaker with shop. "Iron-willed." *Always peculiar,* like other members of family (no insanity in direct line). Became interested in *Christian Science* at 42, but ceased to believe latterly. Climacteric stated to be at 56.

First appearance of mental symptoms variously stated from 58 onwards. At 60, had a *"piano which played without hands."* At 59, bought hens to sell eggs, but the *hens became disfigured, humpbacked, human-faced.* An old horse became *hypnotized* and covered with sores in her presence, only later to be restored to normal. Similar ideas about human beings, commingled with Christian Science doctrines. Her niece willed her a *cashmere kimona* (actually brought for repairs); and this, on obtaining, she kept.

After a year in hospital, sudden change of disposition: disturbed at night, threw water in patients' beds (symbolic baptisms?), seclusive, inaccessible, violence to patients (striking or kicking at approaching persons with an air of great calmness and quietude).

Physique remained perfect until 65, when an epigastric mass, yellowish pallor of skin, nausea and vomiting appeared; and patient died within a month.

Summary of autopsy findings.—Death was due to *general carcinosis* (stomach, pancreas, lungs, peritoneal wall). There was *no emaciation* (thyroid small, soft and flabby, histologically not abnormal with respect to colloid).

Various evidences of chronic disease in trunk: Nephritis, vegetative endocarditis, apical scars of lungs, perisplenitis, myocarditis (edema of ankles) slight aortic and common iliac sclerosis, adhesive pleuritis.

The brain weighed but 1075 gm. and showed *general atrophy,* but perhaps more especially in the post-Rolandic and supra-Sylvian tissues. It is remarkable that *no dilatation of ventricles could be found.* The corpus callosum was of proper proportions. The coronal sections strikingly show the *gaping of sulci in the superior and inferior parietal regions.*

The *gross* distribution of lesions in this brain is suggestive from the standpoint of psychopathological analysis. The imaginative play in the case was unusual, and was commented on early by the

patient herself; and the pseudohallucinational or illusory character of many of the patient's ideas was striking. The increasing and morbid reticence of the patient permitted no analysis of the basis of her catatonic violence ("tranquil" but violent kicking of patients); but remembrance of the *hypnotized horse suddenly broken out with sores* gives a hint of possible mechanism.

It is by no means clear that this case should be dubbed dementia præcox; possibly it belongs in the difficult *presenile* group. Instead of, however, like XV, approaching manic-depressive insanity, the case would appear to approach more a dementia præcox (Christian Science, paranoia, catatonia). Metaphysical delusions (pseudoidealistic conceptions of environment) seem in any event to be associated in this case with parietal-lobe atrophy (little transcortical or subcortical disorder!) which deserves extended histological analysis.

Case XVII.—E. L. (D. S. H. 16674, Path. 1583) was a case regarded as beyond question a *catatonic* example of *dementia præcox,* but with the unusual complication of *glycosuria* (a brother died of diabetes at 12). The patient was of old Vermont stock, had received a partial education, became a domestic, and married at 20. The first child was healthy, the second had spina bifida, the third was a miscarriage followed by nervousness, emotionality, scolding habits; a month later, glycosuria, visual and auditory hallucinations, delusions of poisoning and of influence from a machine, outbursts of excitement, and refusal of medicine and food developed.

Hair of head thin, silky, infantile-feeling. Eyebrows and lashes thin or absent. Axillary and pubic hair scanty. Trophic disorder of finger-nails and toe-nails. Cyanosis of feet and hands, especially of feet.

Pulse during examination often rose to 174.

Cerea flexibilitas marked. *Paræsthesia* described.

Delusions: *Moles* of abdomen described as injection places made by doctors. *"Throat filled up with lead," "Poison in saliva."*

Auditory, tactile and visual hallucinations.

Cerea flexibilitas, impulsive violence, resistivism, refusal to take diabetic diet, furunculosis, finally diphtheria. Death in less than nine months from onset. Wassermann: serum negative.

Summary of autopsy findings.—The autopsy showed a *small hard thyroid,* a *large much injected pituitary gland,* a *small, nodular pancreas.* Microscopic study suggested pancreas as possibly related with the glycosuria. Parts of the pancreas showed apparently normal islands of Langerhans, others few or small ones; in some places polynuclear leucocytes had gathered in considerable numbers in the connective tissue adjacent to islands, but were not often found inside. Whether these leucocytes came in response to necrosis is doubtful.

Death was due to *diphtheritic tonsillitis and pharyngitis.* In addition to the dermal anomalies mentioned above, the autopsy showed a *patent foramen ovale, aortic and common iliac sclerosis,* chronic pleuritis, cholelithiasis, and acute pneumonic areas in each lung.

The brain weighed 1130 gm., possibly a loss or absence of over 100 gm. It lay in an asymmetrical skull, in which the right middle fossa was deeper and broader than the left, to accommodate a *right temporal lobe larger than the left* and of a different (more rounded, blunter) shape. This disparity in size of the temporal regions is exhibited in the coronal sections, which show that the hippocampal gyri do not share in the inequality to any appreciable extent and that the *temporal inequality fades out posteriorly.*

There is a striking difference in the general look of the two hemispheres when viewed from the flank, so much that the casual observer might not readily believe that the two views were taken from the same brain. The sulci of the *right* hemisphere are *more numerous and complex* than those of the *left.* This is everywhere evident, but nowhere more so than in the parietal regions and at the junction of the occipital and temporal regions.

Moderate *presplenial thinning of corpus callosum.* The cranial inequality above mentioned, taken in conjunction with the convolutional inequalities, leads to the hypothesis that this case exhibits many congenital or early brain difficulties, which existed prior to her mental symptoms.

Catatonia and hallucinosis, and also paranoia (early, allopsychic; later somatic) characterized this case. The anomalies seen are largely post-Rolandic and infra-Sylvian.

Case XVIII.—M. W. (D. S. H. 169, Path. 1593) was a case of so-called "*chronic mania,*" which by reason of her development of *catalepsy, cerea flexibilitas* and *neologisms,* might well be regarded as a case of *catatonic dementia præcox.*

Parents peculiar. Patient *solitary and peculiar* at 27. Six months in McLean Hospital at 33. *Denudative, untidy, deluded, incoherent;* admitted to D. S. H. at 37. Delusions bore on *love; wanted to kiss every man she met* (39 years). At 43, *violent,* apparently demented. At 46, *fat,* inactive, lazy. At 55, lump appeared under left mastoid muscle and remained for about six months. At 64, catalepsy, cerea flexibilitas, mutacism. At 68, delusions of grandeur (orders conferred on brother). Neologisms: asked if she slept well, replied, "*Oh! yes, slumosee.*" Asked if food was good, replied, "*Yes, except—coplex—my brother—the great order of—except—mia—plumbo.*" Death at 72 after ten days fever and vomiting.

Summary of autopsy findings.—Death was due probably to septicemia from an ulcerative purulent *cholecystitis* and hypostatic *pneumonia.*

Chronic lesions were: Valvular and parietal *endocarditis, myocarditis,* pleuritis, *perisplenitis, cholelithiasis, nephritis,* aortic and iliac *sclerosis,* fibromyoma of uterus. The liver was *fatty, ovaries cystic,* bone-marrow pale.

Head: *Right-sided frontal endostosis, adherent dura.* Brain weight 1260 gm. (almost normal according to Tigges' formula, 8 x body length, 160

cm. = 1280). The gross appearances, however, indicated considerable loss of weight, in the shape of a *generalized cortical atrophy* (unless the appearances be taken to indicate aplasia?), especially marked in the supra-Sylvian tissues, *superior frontal, precentral,* and *postcentral* areas. The interparietal sulci both anastomose with the Sylvian fissures, and by means of wide bay-like spaces.

It is interesting that the *corpus callosum is not appreciably thinned out,* nor are the ventricles appreciably dilated. The analysis shows that, if we are dealing with an atrophy, it is a rather differential one which does not affect the commissural fibers of the callosum.

Correlations are interfered with by the generalization of the atrophy in this case of 44 years' duration.

Case XIX.—J. K. (D. S. H. 10037, Path. 1602) was a case of *paranoia,* onset at 34 years. A dressmaker, entered W. S. H. at 34, never well since.

Entered D. S. H. at 50 years. Hears enemies talking. Kept awake nights. Lungs torn out by electricity. Has earned a fortune—$100,000 in one year, robbed by a gang. Shocks from a dynamo, followed by bad, heavy feelings in limbs. Gang worked a tube on her, blew gas in nose, producing attacks of coughing and sneezing. Applied to police for protection.

At 52 years, double tertian malarial fever.

At 58 years, *machines are used to tear up back and tear head from body.*

At 61, *hips torn to pieces and hair was torn off.* Later, *fingers were pulled apart, as well as joints and eyebrows.*

Onset of thrombosis and enteritis three days before death at 62 years.

Autopsy 28 hours post mortem.

Summary of autopsy findings.—Cause of death: *Acute enterocolitis, myocarditis.*

Acute lesions: Bronchopneumonia.

Chronic lesions: Atrophy of intestinal mucosa; aortic sclerosis, internal carotid arteriosclerosis, tricuspid valvular sclerosis; chronic nephritis; chronic splenitis; cholelithiasis; cystic right ovary; favus of vertex; chronic pachymeningitis; arcus senilis; grooving of calvarium for vessels; absence of teeth.

Nervous system: Brain weight 1315 (excess 59 gm. by Tigges' formula, 8 x body length, 157 cm.).

The brain of this case was distinguished by a marked diffuse chronic *pial thickening* (especially over vertex) and by a marked degree of *internal hydrocephalus* (such as would hardly have been suspected from the brain's external aspect or its weight of 1315 gm.). There was only slight basal cerebral arteriosclerosis. The grossly evident *posterior-column degeneration* of the spinal cord might also give rise to suspicions as to the integrity of the brain itself (or even suggest a luetic origin for the total picture). The *posterior half* of the main part *of the corpus callosum* was slightly *thinner* than normal. The frontal polar gyri are richly *subdivided,* especially on the orbital surface (also the *lingual gyri*). A double commissure was discovered on dissection.

Possibly a case of *paraphrenia phantastica* (compare Cases IV, VII). Like IV and VII, XIX shows generalized disease, but of a different genesis (leptomeningitis and internal hydrocephalus).

Case XX.—Z. T. (D. S. H. 6334, Path. 1603) was a case of *"secondary dementia"* with symptoms dating back to 33 years or earlier. At first, paranoidal (homicidal, suicidal, "poisoned") and unusually dangerous. In hospital, given to impulsive violence—knocked down and stamped upon a patient because "he would not dress and had over some of his talk to him."

Imagination unusually varied: expansive ideas about wealth, mines owned, relation to royalty. Would often state that he was some notable person. *Had a job polishing off the moon. Moon floats in the ocean like a bubble in the day, reached by man-of-war.* Drew *"marine views"* with colored water got by soaking colored paper. *"Bushes growing inside of body," "daughter inside," "1000 years old."*

Glycosuria at 50. Death at 67.

Autopsy 22 hours post mortem.

Summary of autopsy findings.—Cause of death: *Cerebral hemorrhage.*

Acute lesions: Acute splenitis, acute bronchitis.

Chronic lesions: Obesity; coronary, internal mammary, and cerebral arteriosclerosis; mitral and aortic valvular endocarditis; chronic nephritis; fat-replacement in pancreas; arcus senilis; chronic thyroiditis.

Brain weight (with hemorrhage) 1550 gm.

Death in this case was due to an extensive *right-sided cerebral hemorrhage.* (Cf. Case XXII (1616).) It is clear, however, that a marked chronic *pial thickening over the vertex* and a marked degree of *internal hydrocephalus* antedated the hemorrhage.

Perhaps the most striking feature of the brain is an anomaly of the *left superior temporal gyrus* remarkably similar to that of Case III (1319). It is interesting that the coat of pial thickening spreads down from the vertex *over the left opercular region* (not markedly on the right) and is especially marked *over the left frontal operculum* (where stripping off the membrane reveals a somewhat deep-lying gyrus, perhaps presenting a similar type of anomaly to that of the above-mentioned superior temporal anomaly).

Investigation of this case histologically might seem unprofitable were it not for the somewhat similar anomaly of Case III (1319), in which hemorrhage did *not* occur and in which the age at death was 31 instead of 60. It will be noted, however, that there was also in Case III (1319) an internal hydrocephalus, but more marked on the left side.

This case, like XV, seems to show little evidence of either catatonia or hallucinosis, and is marked by delusions of grandeur and fantastic autopsychic delusions. The brain shows frontal opercular, and to some extent parietal opercular, lesions; but,

unlike XV, this case shows anomaly of superior temporal convolution.

Case XXI.—A. G. (D. S. H. 14001, Path. 1615) was a lady's maid, born in France, queer after 34 years of age. Ideas of reference (auditory hallucinosis) at 45. Strong feelings in forehead (a girl had put chloroform there, she said). *Chloroform and powders smelled. Voices* ("bad woman," etc.) *heard.* Hyperreligiosity. Wrote innumerable prayers in archaic French and bound them in books of her own manufacture. *"Absences of thought."* One seemingly hysterical attack, while menstruating, followed by hypalgia, incontinence of urine, muscular weakness, and later by amnesia.

Summary of autopsy findings.—Cause of death: *Laryngeal diphtheria.*

Acute lesions: Dermatitis of face, hands, wrists, genitalia.

Chronic lesions: Periappendicitis and generalized adhesive peritonitis; obliterative pleuritis, right; cholelithiasis.

Nervous system: Brain weight 1170 gm. (8 x body length, 140 cm., would equal 1120).

Attention is at once drawn in this case to the two *inferior parietal regions,* which are both (and more notably the left) subject to a kind of general retraction from the main contours of the surrounding parts. This retraction appears to center about the *interparietal sulci* in their *posterior* portions. Over these sulci and the inferior parietal lobules there is a slight *pial opacity,* without demonstrable thickening and with borders playing off vaguely into the adjacent normal-looking pia mater.

There is a marked rather suddenly appearing *thin place* in the posterior quarter of the main part of the *corpus callosum.*

The impression is also conveyed of a slight to moderate degree of *atrophy of the opercular region,* more marked on the left side. Internal hydrocephalus, if present, is but slight in amount.

Hallucinosis seemed to command the paranoia of this case, and it is a question whether the long archaic French prayers were not dictated by voices. The paranoia seems on the whole "impressional" or ideomotor, and therefore consistent with the (inferior) parietal and opercular atrophy.

Case XXII.—E. A. (D. S. H. 1227, 2009, Path. 1616) gave first symptoms at 26: melancholia, after business reverses; fainting fits; idea of servant girl as a Boston lady in disguise (attempted to enter her room on the ground that he was married to her). Improved somewhat in hospital, but returned at 27. At 29 spoke of God's commands. Assumed a German accent. Occasional violence, surliness, abusiveness. At 33, muttering and gesticulating. At 35, rhythmical motions with one hand. At 45, walked with head bent back and turned to one side. Mutism for years. Shock at 56 (transient spastic right-sided paralysis, clonus, Babinski). A diagnosis of organic dementia (luetic) was then considered. After being mute for years, patient suddenly spoke, objecting to a throat culture being taken. Death at 59.

Summary of autopsy findings.—Cause of death: *Cerebral hemorrhage.*

Acute lesions: Hemorrhages in floor of fourth ventricle, pancreas, stomach, right sclera; early ulceration of colon; large mesenteric lymph-node; bronchopneumonia.

Chronic lesions: Aortic sclerosis; pleuritis, right side; periappendicitis; pigmented moles.

Nervous system: Brain weight 1220 gm. (8 x body length, 164 cm. = 1312), a reduction despite the hemorrhage of the right hemisphere.

Like Case XX (1603), Case XXII (1616) died of *right-sided cerebral hemorrhage.* Like 1603 also, the case showed *internal hydrocephalus.* There was, however, no marked degree of pial thickening.

Curiously enough, there is also in this case an approach to the *left-sided superior temporal anomaly* shown in Cases II (1317), III (1319), XX (1603). This case appears to suggest a vascular origin for the anomaly, in that numerous apparently serpentine vessels were found coursing over the opercular regions.

Case XXIII.—L. S. (D. S. H. 11176, Path. 1622) was a case of *dementia præcox.* Onset at 22 years. Entered McLean Hospital at 22. Love affair. Told she was making herself and family conspicuous, wept all night. Moody, praying, said strange things, taciturn, apathetic, suicidal threats. Heard father calling her. Disoriented. Violent and profane. Occasionally laughed and sang. Muscular spasms and clonic spasms when asleep.

Admitted to D. S. H. at 24 years. Oriented. Depressed. "Its a pity she is so wicked." "My body is vile." "Tormented with voices, ashamed of everything." Insight. Violent outbreaks, ideas of persecution and unreality. Somatic ideas (*head and brain of sawdust*). Lay about on couch, hair down. At 29 to 30 years, mute and resistive, constantly hallucinated, "men in her room to lead her astray," oriented, unsocial. Later denudative. Four days before death at 34 years, developed lobar pneumonia.

Summary of autopsy findings.—The brain of this case has a simple pattern. (Compare, *e. g.,* the richness of sulcation in the *orbital and lingual gyri* of Case XIX (1602).) The *parieto-occipital fissures* are deeply sculptured. The sulcus which penetrates the *left supramarginal gyrus* seems also unduly deep. The *sulci separating postcentral gyri from inferior parietal lobules* are continued into the fissure of Sylvius, forming a kind of ascending middle branch to each; these are also *deeply hollowed out,* as also are the ascending branches of the Sylvian fissures. There is a slight chronic *leptomeningitis,* most marked over the mouth of the *left Sylvian fissure.* However, one gets the impression that the *superior parietal* atrophy (or aplasia?), as represented by the deepening of the parieto-occipital fissures, is the most marked lesion of the brain.

The corpus callosum strikes one as of good proportions, and *fails to show thinning out posteriorly.*

At first paranoidal, later catatonic, this case offers difficulties in correlation. Hallucinosis was prominent at various periods in the course, but there is little to show in the gross brain. Therefore, since the very pronounced atrophy is mainly above the fissure of Sylvius (microscopic examination, it is true, shows considerable change in the superior temporal gyrus, but the left supramarginal gyrus leads all other areas in extent of atrophy from the microscopic point of view), we may need to be content, so far as gross correlations go, with a parietal correlation for the catatonia.

Case XXIV.—L. L. (D. S. H. 16063, Path. 1625) had a paternal uncle insane (type unclassified), and was always seclusive. Married at 22 (four children), lost husband at 30, worked hard. At 34 was unconscious from a fall, and four months later became strange and talked strangely (ideas of reference; disconnected letters; catatonic stupor 10 days, two days lucid, again stuporous or catatonic till death at 39). Denudative, resistive, negativistic, verbigeration, untidiness, assumption of fixed positions, at times cerea flexibilitas, silent with doctors.

Summary of autopsy findings.—Cause of death not clear.

Acute lesions: Cervicitis, injection of ileum.

Chronic lesions: Emaciation; large mesenteric lymph-nodes; chronic nephritis, with focal areas of sclerosis; gastroptosis; bones brittle.

Nervous system: Brain weight 1190 gm. (1200 gm. by Tigges' formula). Slight leptomeningitis along sulci.

The most striking feature of the brain of this case is the somewhat sharply marked *thinning of the posterior half of the main portion of the corpus callosum.* There is also a moderate *dilatation of the posterior halves of the bodies of the lateral ventricles.*

The *convolutional pattern* of the brain *gives the impression of simplicity,* except upon the inner face of the occipital regions, which is either atrophic or aplastic, judging from the gross appearances.

Especially simple in construction appears to be the temporal lobe of each side. There is another example of anomalous Sylvian branching, reminding one, more especially on the left side, of conditions in Case XXIII (1622). The area of Broca and the co-ordinate right-sided area show rather deeply hollowed sulci.

This almost exclusively catatonic case is of note in that all the pre-Rolandic tissues fail to show appreciable lesion, whereas a somewhat marked posterior dilatation of the ventricles, thinning of the posterior part of the corpus callosum, and occipital atrophy or aplasia (inner surfaces) and simplicity of the temporal lobes emphasize the sensorial and supersensorial nature of the lesions. (Microscopically, all areas examined showed marked satellitosis.)

Case XXV.—L. P. (D. S. H. 16779, Path. 1634) was a case of *dementia præcox,* onset at 17 years. Father insane, dementia præcox. At 17 *feared Catholics. Men creeping in through window, devils and negroes.* Ideas of persecution.

Admitted to D. S. H. at 17 years. Restless, talking incoherently and impulsively singing. Motions quick and impulsive. Mannerisms, tube-fed, untidy.

Cerea flexibilitas 18 days after entrance. Hums to herself, recognizes mother but does not talk to her.

Symptoms thought to be those of pulmonary tuberculosis two months before death at 18.

Summary of autopsy findings.—Cause of death: *Tuberculous abscesses of left lung.*

Chronic lesions: Chronic obliterative pleuritis, left.

Brain weight 1270 gm. (excess 142 gm. by Tigges' formula, 8 x body length 141 cm.).

This brief case of catatonia, having a brain practically normal in gross (with a suggestion of ventricular dilatation posteriorly), was studied microscopically in advance of the total series, and shows marked characteristic changes everywhere. Correlations of lesions to particular symptoms are accordingly impossible.

Perhaps this is a case in which tuberculosis has something to do with the process. At least the tuberculosis and the mental process appear to have run their course together.

V. General Discussion.

I am disposed to believe that the present work, confirmatory as it in general is of my former work of 1910, goes very far toward *placing dementia præcox in the structural group of mental diseases.* Previous work of several authors had left much to be desired in respect to proofs of the structurality of this disease, because reliance was placed on *microscopic* examination and the observation of various cell changes, some of which might well be reversible or terminal or intercurrent, or even agonal. Even the initial conclusions of Alzheimer concerning deep-layer cortical gliosis in catatonia (1897) are now replaced by Kraepelin (1913) with an emphasis on changes in *supra*-stellate layers. Nor did any author seem to reckon with the fact that such microscopic changes as are sometimes adequately described must inevitably leave, as a rule, some macroscopic trace, if the disease had lasted a long enough time (say three months, more or less). Accordingly, in

the absence from the literature of all systematic gross brain study in dementia præcox, at least of such study in any large series of cases, I undertook to fill the gap and have fortunately been able, through grants from the Massachusetts State Board of Insanity, to execute the work with the aid of systematic brain photography. Careful and systematic autopsy protocols, brain photographs systematically taken, and microscopic sections in all cases of possible doubt as to gross diagnosis, have formed the basis of the work on the structural side.

Before considering the data, it may be inquired whether the cases presented are really cases of dementia præcox, since *diagnosis* in this disease or disease group is admittedly difficult. The condensed histories given in the text speak for themselves. Possibly some cases of manic-depressive insanity of mixed type may have crept into the series; if so, the anatomical correlations could only be lowered by our diagnostic error. There may be instances of imbecility in the series, but none, I believe, without a strong dementia præcox coloring; if such diagnostic errors were plentiful, it is likely that the correlation with agenesic lesions would become too high, and the hypothesis of acquired hypoplasia (otherwise acceptable) would correspondingly suffer. In any event, I believe, there is no false bolstering of the high anatomical correlation by means of cases having acquired lesions obviously too recent or otherwise theoretically irrelevant to explain the symptoms.

Among the 25 cases systematically studied, there were but two without evidence of cortical atrophy. These two (VIII, of three weeks' duration, and XXV, of two months' duration) may not have been cases of dementia præcox (since it is doubtful whether the diagnosis can be made safely in so brief a period); but on the whole it seems desirable to count them as such. One of them (VIII) had a decidedly asymmetrical face and brain (as well as other somatic anomalies). Both cases showed microscopic changes adequate to account for many symptoms. Indeed, the universality of microscopic changes in Case XXV would lead to the idea that this case was proceeding towards a general mild atrophy (should survival have permitted). The brief duration of these cases probably accounts for the absence of macroscopic evidence of brain disease.

What is the *nature of the lesions* found? It seems that they

might be (a) *acquired* by the individual or (b) *congenital;* and evidence in each of these directions had been supplied from the work of 1910. But, if acquired, the lesions may still have arisen quite out of relation to the development of symptoms. Thus, they may have been *acquired early* in life and thus have been tantamount to congenital lesions, or at all events extinct so far as symptom-production is concerned. Or, on the other hand, they might come to be *acquired late* in the course of the disease, and be either chronologically *secondary* to the disease or even *incidental* and adventitious results of something quite apart from the disease. If *congenital,* the lesions might be examples of agenesia in the sense of a circumstance under which certain cells altogether fail to be developed, or they might be examples of *aplasia or hypoplasia* in the sense of a failure of cells which have been quite properly laid down to progress in the normal direction, *e. g.,* to the normal size, or with all the normal appendages, or with normal powers of resistance, or the like. Besides these possibilities, conditions like *hydrocephalus* might occur (see below), in which anomalous results would follow upon cell readjustments entailing neither agenesia nor aplasia. And, again, it would be conceivable that *acquired skull distortion, abnormal cerebrospinal-fluid pressure, abnormal blood pressure,* and cognate conditions (such as have been recently discussed by my colleague, H. M. Adler), should bring about abnormal conditions without the loss of a cell, at least in early phases of the particular abnormal physical state. And combinations of such acquired and congenital features might occur, and some of them were previously noted to occur (1910).

Whatever the general outcome of the inquiry, it might well be that *loss of brain weight* should eventually supervene. In 1910 I found a slight loss of two per cent; the present series shows such marked variations from case to case that conclusions must be withheld, although there are striking individual instances of loss.

Heart, liver, and kidneys show proportionally more loss of weight than does the brain. The kidneys are more atrophic in this series than in the series of 1910. Excluding certain cases with long-standing splenomegaly, the spleen also shows a tendency to low weight.

How far are the lesions and anomalies of this series interpretable on the hypothesis of dementia præcox as incidentally organic?

In the first place, the lesions and anomalies are practically constant in occurrence in the new series (23 out of 25 cases), and there is important evidence of similar focal lesions in the great majority of all cases so far examined (45 out of 50 cases). We are left with a small residuum of cases in which no registration *in the gross brain* has occurred. Microscopic examination, so far as we can rely on it, indicates a tendency to similar changes in several of the cases in the "normal-looking" residuum. When we remember the elusive nature of many important gross lesions and anomalies (so often missed in routine autopsies), when we bear in mind that the patient must survive a certain period in order to yield gross evidence of brain disorder at all, and above all when we remember the difficulties in diagnosis in this group, we cannot be surprised at our not forthwith arriving at 100 per cent of gross lesions and anomalies in this series of cases. (As to diagnosis, it should be added that all cases have been included which could by any possibility have been regarded as instances of dementia præcox, and it is safe to say that the percentage of gross lesions has not been artifically increased by any form of exclusion of cases. In particular, I have *not* omitted those difficult cases in which discussion at present rages concerning the differential diagnosis of dementia præcox and manic-depressive insanity of *mixed type*.)

But, though it be granted that our findings are practically constant, it might still be that they are only incidental, much as broken glass might be constantly or almost constantly found after a banquet with wine.

Logical formulæ do not provide an answer to this question. In the particular form in which Meyer has expressed the notion of "incidentally" organic lesions, one sees a suggestion of Edinger's *Ersatztheorie*. Cells, working in such and such abnormal ways, are eventually subject to metabolic changes and final breakdown. Supposing this to be true, and supposing the process to be initiated by some non-cellular event (*i. e.*, by some event that does not interfere at once with normal cell-functioning), still the progress of cell-functioning is by hypothesis sooner or later disturbed. Thus, if I am correct, the disease might be regarded as having two phases—a non-cell-injuring phase and a cell-injuring phase.

I am far from certain that this issue can now be resolved, and I see the tactical advantage of holding to a non-cell-injuring phase

in dementia præcox (*therapeutic optimism!*). But, if the issue is constantly or even very frequently a phase of cell-injury, then it is plain that something of peculiar potency must lurk in the non-cellular events that initiate dementia præcox; and, what is still more important, it is obvious that the cell-injuries themselves must have an important bearing on the progress of the disease. Thus I am loth to admit that there can be "incidental" changes in the brain which will not be of profound significance to its function. The brain is so labile an organ, and many parts of it phylogenetically so new, that even small changes must be of great consequence to it.

Accordingly, in exchange for the tactical advantage of a provisional therapeutic optimism, I would prefer the strategic advantage of a research point of view which should study intently the cellular results (if they are results), the cellular progress, and the cellular causes (if there are any) of this disease.

As a last word concerning this problem, I would say that I think there are reasons for supposing that there may be no fundamental difference between the so-called structural ("organic") and the so-called functional points of view. I believe that some day it may transpire that events apparently non-cellular are really cellular, or, otherwise expressed, there may be but one series of events, some part of which is conveniently termed psychical and some part physical. This is no place in which to discuss this view; I need only say, in addition to already published remarks, that the new view will probably not serve either to identify the psychical with the physical or *vice versa*.

Granting that a new ontology on such lines should enter to replace the parallelistic or interactionistic ontologies which now prevail, we should be forced, however, not to regard the changes we find *sub specie structuræ* as either causative of or incidental to the changes we find *sub specie functionis*. All relevant and pertinent changes would, to the new point of view, assume their proper place in some higher genus of things and events.

I wish to lay no particular stress on these ontological points here, and merely submit, concerning the concept of the "incidentally organic," that "*incidental*" should not receive the connotation of (a) *infrequent,* since the changes are certainly not infrequent, or (b) *unimportant,* since, after they occur, it is hard to

see how such changes could fail to be of importance. It is possible that the term "incidental" is intended by Meyer to connote a *secondary* nature for these changes; if the changes are secondary but constant, it would be hard to deny them a measure of importance. Possibly, however, "incidental" is meant to be taken as *secondary in point of time* (this is suggested by Meyer's term "endogenous break in compensation"), and this meaning is the one which I should take most seriously. Here would lodge the most momentous issue as to facts. *Does the process of dementia præcox begin with a non-cell-injuring phase?* That seems to me to be the best-defined issue of Meyer's attempt to regard changes found in dementia præcox brains as incidental.

This leads to a consideration of what I understand to be August Hoch's attitude to the anatomical findings in dementia præcox. I understand him to believe that they represent either an agenesia or the effects of such agenesia of the brain. Of course the omitted (agenesic) elements are few, and the resemblance of dementia præcox to higher grades of imbecility is often more apparent than real.

I myself hold rather that such findings as we now have indicating maldevelopment (and there are surely many such) indicate rather an aplasia than an agenesia, rather an interference with development of cells laid down than an interference with their laying down. But, of course, an early aplasia may go far to simulate an actual agenesia, so that the issue is in a particular case often never to be resolved.

In the 1910 series, I studied from this particular point of view 15 cases, of which 14 yielded features which I regarded as "acquired" (the 15th case happens to figure also as Case IV (1335) of this series, and suggests on further study also an acquired atrophy of the occipital, but especially of the temporal, regions). But at the same time, 8 of the 15 cases had more or less pronounced features indicating early hypoplasia or (possibly in some cases) agenesia.

The comparative infrequency (8:15) of these developmental changes might lead one to underestimate their value as an index, and they might be dismissed in one of the senses of the term "incidental." But I am inclined to hold that further study will increase rather than diminish their number.

The following table divides the pertinent lesions found into provisional groups as acquired and developmental. For the purposes of the present discussion, I have pushed into the developmental column all cases that could by any interpretation belong there from the anatomical point of view. I have, for example, put all the examples of chronic internal hydrocephalus in that group, although I do not feel that they all belong there. It is also doubtful whether all the cranial asymmetries are developmental.

TABLE XI.

Acquired and Developmental Lesions.

NO.	ACQUIRED.	DEVELOPMENTAL.
I (1297).	Left cerebral atrophy.	
II (1317).	Left Sylvian atrophy.	Left superior frontal hypoplasia; hippocampal hypoplasia?
III (1319).	Slight basal leptomeningitis.	Left temporal hypoplasia, especially superior temporal.
IV (1335).	Temporal and occipital atrophy.	Microcardia, microhepar, small brain.
V (1383).	Cerebral atrophy, especially pre-Rolandic, right. Thinning of corpus callosum.	
VI (1413).	Frontal atrophy (slight) (see opposite also).	Callosomarginal anomalous plication (but early encephalitis?).
VII (1472).	Cerebral atrophy, or...	Cerebral hypoplasia (agenesia?).
VIII (1487).		Patent foramen ovale, persistent thymus, facial asymmetry, asymmetry of brain.
IX (1491).	Paracentral atrophy?	Chronic internal hydrocephalus.
X (1507).	(Brain metastasis of carcinoma) frontal atrophy?	Bilateral temporal agenesia (undue simplicity of plication).
XI (1509).		Patent foramen ovale, facial asymmetry, fetal convolutions of spleen; moderate chronic internal hydrocephalus.

NO.	ACQUIRED.	DEVELOPMENTAL.
XII (1518).	Sclerosis of right callosomarginal gyrus.	Probably the lesion opposite is in some sense hypoplastic; small right cornu ammonis; chronic internal hydrocephalus; left frontal aplasia.
XIII (1540).	Superior frontal atrophy (aplasia?) left angular atrophy.	
XIV (1571).		Cranial asymmetry.
XV (1574).	Atrophy of frontal and parietal operculum; thinning of corpus callosum.	Cranial and cerebral asymmetry.
XVI (1575).	General cortical atrophy, especially post-Rolandic and supra-Sylvian.	
XVII (1583).		Marked cerebral asymmetry of gyri.
XVIII (1593).	General cortical atrophy; especially supra-Sylvian.	
XIX (1602).	Marked chronic leptomeningitis.	Chronic internal hydrocephalus (acquired?).
XX (1603).	Frontal and parietal opercular atrophy; chronic leptomeningitis.	Chronic internal hydrocephalus; anomaly of left superior temporal gyrus.
XXI (1615).	Opercular atrophy, more marked, left.	Retraction of both inferior parietal regions.
XXII (1616).		Chronic internal hydrocephalus; left superior temporal anomaly.
XXIII (1622).	Chronic leptomeningitis.	Superior parietal aplasia (atrophy?).
XXIV (1625).	Thinning of corpus callosum.	Partial chronic internal hydrocephalus.
XXV.	Negative in gross.	Negative in gross.

The result of this tabulation is to show that 19 of the 25 cases might possibly be regarded as in some sense maldevelopmental. An impartial witness to whom I submitted the data thought that surely 14 of these 19 were true instances of maldevelopment. If the eight cases of chronic internal hydrocephalus are all acquired and not developmental, then we still have outstanding 11 cases of maldevelopment.

CLINICAL CORRELATIONS WITH CHRONIC INTERNAL HYDROCEPHALUS.

The fact that almost one in three of our random series of dementia præcox brains exhibited more or less well marked partial or general chronic internal hydrocephalus, forms a novel and many-sided problem.

The list is as follows:

TABLE XII.

CORRELATIONS WITH INTERNAL HYDROCEPHALUS.

No.	Sex.	Age.	Classification (Kraepelin, 1899).	Duration.	Nature and degree of hydrocephalus.
III (1319).	F.	31	Paranoia (imbecile?).	8 yrs.	Moderate, more of left lateral ventricle.
V (1383).	F.	62	Catatonic.	Years.	Marked, especially in mid portion.
IX (1491).	F.	53	Catatonic.	Years.	Moderate, equal on both sides.
XI (1509).	M.	58	Catatonic.	37 yrs.	Moderate, equal on both sides.
XII (1518).	F.	45	Catatonic.	29 yrs.	Slight general, more marked in two posterior cornua.
XIX (1602).	F.	62	Paranoic.	28 yrs.	Marked, equal on both sides.
XX (1603).	M.	67?	Paranoic.	34 yrs.	Marked, probably equal (marked by complicating hemorrhage).
XXII (1616).	M.	49	Catatonic.	23 yrs.	Moderate, equal on both sides.
XXIV (1625).	F.	39	Catatonic.	5 yrs.	Moderate of posterior halves of bodies of lateral ventricles.

The meaning of this hydrocephalus may be taken up from various points of view.

Age is not a specially powerful factor. Four of the cases were under 50 at death, five over 50.

Duration of symptoms is suggestively long. The briefest case had symptoms for five years (one is of unknown duration, probably decades). No case of brief duration exhibits hydrocephalus

On the other hand there are fairly numerous cases of great duration without hydrocephalus. Examples are:

No.	Sex.	Age.	Classification.	Duration.	Area involved.
*I (1297).	F.	47	Paranoic.	27 yrs.	Atrophy confined to cortex.
II (1317).	F.	44	Catatonic.	21 yrs.	Frontal and temporal hypoplasia.
VII (1472).	F.	55	Paranoic.	27 yrs.	General brain atrophy (or aplasia).
X (1507).	F.	60	Paranoic.	36 yrs.	Frontal and temporal atrophy.
XVIII (1593).	F.	71	Catatonic.	44 yrs.	General cortical atrophy
XXI (1615).	F.	58	Paranoic.	24 yrs.	Inferior parietal atrophy (aplasia?).

Thus there are six cases of long duration (over 20 years from first recorded symptoms) which did *not* show hydrocephalus. IV (1335), XV (1574), and XXIII (1622) are three others of fairly long duration (20 or 14 years, decades? and 12 years respectively) which also did *not* show hydrocephalus.

It is obvious, accordingly, that neither the age of the patient nor the duration of the symptoms possesses any regular relation to the appearance of hydrocephalus. It is true that the two most pronounced instances were in the oldest cases—XIX (1602) and XX (1603)—and in cases of long duration (28 years and 34 years respectively). On the other hand, two cases of shortest duration—XXIV (1625) (5 years) and III (1319) (8 years)—also showed moderate hydrocephalus, albeit partial (more on left side and in posterior halves of bodies of lateral ventricles respectively). But in one case of 29 years' duration, with death at 45—XII (1518)—the hydrocephalus was also more marked in the two posterior cornua.

It remains to be inquired whether the symptoms of the hydrocephalus cases presented anything in common. The group is in point of fact an uncommonly catatonic group. Although three cases are tabulated as paranoic, in point of fact two of these showed various catatonic or allied features (III, attitudes, resistivism,

* This case was variable, cyclothymic, and it is a question how much of the period of 27 years was occupied by processes which could affect the brain.

refusal of food; XX, impulsive violence), and the third (XIX) had a great variety of somatic delusions which might well indicate effects wrought in the postcentral gyrus as well as elsewhere. But whether the hyperphantastic somatic delusions of Case XIX are to be regarded as in some sense a catatonic equivalent (heavy feelings in limbs, various delusions of being rent apart), the other cases are all more or less pronouncedly catatonic.

But the group is also somewhat given to hallucinations (III, visual, gustatory (olfactory?); XI, auditory; XIX, auditory; XX, poison (hallucinatory?); XXII, auditory). The cases without hallucinosis were as follows: V and IX, decidedly inadequate history (ideas of poisoning); XII, slight general hydrocephalus, but more marked in posterior cornua; and XXIV, moderate hydrocephalus, confined to posterior halves of bodies of both lateral ventricles. The hallucinatory tendency is therefore stronger when the hydrocephalus is marked and general.

The hydrocephalic group appears not to be so much given to delusions as to catatonia or even to hallucinosis. However, there were three cases tabulated above as paranoic (one of these, Case III, on rather insufficient grounds). Of these three, two were cases in which hyperphantastic delusions, often somatic in content, occurred. It would be my theoretical desire to correlate such hyperphantasia on general grounds rather with lesions or conditions of the posterior association center than with such in the anterior center. There seems to be in these cases of hyperphantasia no necessary development of any morbid or unusual reaction to the delusions expressed. The victim of hyperphantasia is often in a state of passivity like a spectator at the play, and of such a patient it can hardly be said that his attitude or conduct (which we are here attempting to correlate with frontal conditions) is in any respect abnormal.

On the whole, therefore, if I interpret along my previously suggested lines, I see far less evidence of morbid frontal-lobe action (motivated attitude and conduct) than of morbid parieto-temporal action (catatonia and hallucinosis). It is interesting in this regard that there were two instances of hydrocephalus of a more posterior locus, from which it might be thought that for some reason the dilatation is apt to begin there as at a point of lesser resistance to expansion. Both of these local posterior

hydrocephalus cases were markedly catatonic, and neither showed either hallucinosis or delusions (except that XXIV had some ideas of reference).

This problem of the comparative immunity of the hydrocephalus cases from delusions can also be approached from the anatomical side (if the frontal-paranoia correlation be for the moment taken for granted) by inquiring what effects have been wrought by the hydrocephalus cases upon the frontal lobes. These correlations may be studied in detail in the table of Gross Anatomical Correlations with Paranoia (Insane Delusions).

No effects of a gross anatomical nature in the frontal region, whether caused by the hydrocephalic process or not, are to be seen in III, IX (except a slight mesial thinning posteriorly), XIX, XX, XXII, XXIV (except a question of atrophy or aplasia of *inferior* frontal gyri). Of the three cases which do show frontal-lobe lesions, V (moderate frontal atrophy) and XII (left-sided frontal aplasia) had also been inferred in two instances, on clinical grounds, to have shown delusions; whereas in the third (XI), history is lacking. On the whole, therefore, it seems safe to say that, in this group at least, hydrocephalus bears far less hard on the frontal association centers than on the parietotemporal association centers.

Accordingly, if our general contentions be at all correct, the hydrocephalic cases of dementia præcox will be found more often catatonic or hallucinated than deluded (except that hyperphantasia occurs with hydrocephalus).

I assume that, whatever be the conditions provocative of hydrocephalus, it is likely that the process will vary in degree from time to time. It is quite consistent with this conception that the cases of this group should show themselves (as an inspection of the histories will prove) more than usually active and mutable. I also find a tendency to attacks and exacerbations marked in three cases (XI, six attacks; XII, four or more attacks or exacerbations; XXI) and indicated in others.

The first hypothesis which the anatomist would have concerning the cause of the hydrocephalus would be that it is a hydrocephalus due to brain atrophy.

The weights were:

III.	1250 gm.
V.	1060 gm. (60 minus, Tigges).
IX.	1330 gm. (50 plus, Tigges).
XI.	1265 gm. (100 minus, Tigges).
XII.	1385 gm. (105 plus, Tigges).
XIX.	1315 gm. (60 plus, Tigges).
XX.	1550 gm. (with hemorrhage).
XXII.	1220 gm. (92 minus, Tigges).
XXIV.	1190 gm. (10 minus, Tigges).

There is on the face of these figures no convincing evidence of brain atrophy in these hydrocephalic cases, since there is no case which by Tigges' formula has lost more than 100 gm. However, the technical fact must be remembered that the dilated ventricles contain more fluid than normal ones and that, if the brain is weighed at once without thorough drainage (as I am sure has happened at times), the scales may register 100 to 150 gm. of fluid additional to the normal amount (sometimes set at 125 gm.) and thus give a deceptively high value to the brain weight. Accordingly there may be more atrophy than one would suppose from the figures.

In the above remarks I have proceeded as if it were quite likely that the hydrocephalus is acquired. If congenital, we might expect to find various other evidences of aplasia. Case III indicates such aplasia of the left temporal region, probably closely associated with the more marked left-sided internal hydrocephalus. Case IX showed a striking difference in general shape of the two hemispheres (cranial malformation or alteration?). Case XII showed what I have termed above "a right-sided hippocampal aplasia in relation to a left-sided frontal aplasia" (there were also appearances in this case consistent with the hypothesis of an early encephalitis). On the whole, therefore, it would seem that there is not much evidence of an association of the hydrocephalus with congenital features.

CORRELATIONS OF TEMPORAL-LOBE LESIONS WITH AUDITORY HALLUCINOSIS.

Following is a table designed to bring out sharply the presence or absence of anatomical correlations with hallucinations of various types (although mainly auditory). The table presents all 25 cases, including 13 cases (IV, V, VIII, IX, X, XII, XIV, XV,

XVI, XVIII, XX, XXIV, XXV) *without* history of hallucinations. In the anatomical column (on the right) have been placed, for these 13 supposedly negative cases, all lesions which might have been thought able to favor hallucinosis. The table can therefore be used in two ways, to try the strength of (a) the correlation between hallucinosis and appropriate cortex lesions (*e. g.*, auditory hallucinosis against temporal-lobe lesions), and (b) the correlation between such lesions and hallucinosis.

The results of the analysis are summed up *infra*.

TABLE XIII.

GROSS ANATOMICAL CORRELATIONS WITH HALLUCINOSIS.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
I (1297).	Auditory (reviling, "not real," also noises in head).	Peripheral deafness, bilateral; area of softening, right second temporal gyrus.
II (1317).	Auditory.	Anomaly (with atrophy? or aplasia?) of left superior temporal gyrus. Left temporal lobe poorly supplied with white matter.
III (1319).	Visual?, gustatory?, auditory?	Chronic internal hydrocephalus, left superior temporal anomaly, and poor supply of white matter to left temporal lobe (cf. II).
IV (1335).	*No evidence of hallucinations;* fantastic delusions (sensorial in origin?).	General hypoplasia (with atrophy?), more marked behind Rolando and below Sylvius.
V (1383).	*No evidence of hallucinations.*	Temporal and parietal (except postcentral) regions not especially involved. Chronic internal hydrocephalus, especially of mid portion.
VI (1413).	One episode of auditory hallucinations; none later.	No evidence of gross temporal-lobe lesions.
VII (1472).	Early brief period of possibly auditory hallucinations, or possibly ideas of reference construed as hallucinations; later probably *Gedankenlautwerden.*	General hypoplasia, or more probably atrophy of brain; gyri on both sides of Sylvius slightly more atrophic (hypoplastic) than other gyri.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
VIII (1487).	*No evidence of hallucinations* (total course three weeks).	No sclerosis or atrophy elsewhere. Asymmetry of brain (three weeks' duration).
IX (1491).	*No evidence of hallucinations.*	No sclerosis or atrophy in appropriate regions (paracentral atrophy (aplasia?)). Chronic internal hydrocephalus.
X (1507).	*No evidence of hallucinations.*	Temporal regions simple in plication; moderate atrophy of temporal lobes.
XI (1509).	Auditory hallucinations inferred from some of patient's attitudes.	Atrophy or aplasia of anterior portion of each superior temporal gyrus. Moderate chronic internal hydrocephalus.
XII (1518).	*No evidence of hallucinations.*	No appropriate lesions or anomalies. Slight chronic internal hydrocephalus.
XIII (1540).	Auditory hallucinations surmised from some of patient's acts.	General mild atrophy and sclerosis of brain; no special emphasis in temporal regions, although the plication of the *right* temporal area was the simplest found in all cases examined in this series.
XIV (1571).	*No evidence of hallucinations;* somatic delusions probably on a peripheral basis.	No appropriate lesions or anomalies. Cranial asymmetry, possibly acquired, and possibly related to somatic delusions.
XV (1574).	*No evidence of hallucinations.*	No appropriate lesions or anomalies.
XVI (1575).	No evidence of true hallucinations; remarkable pseudohallucinations ("overimagination").	No temporal-lobe lesions or anomalies. General cortical atrophy, but most marked behind Rolando and above Sylvius.
XVII (1583).	Visual, auditory and tactile hallucinations.	Left-hemisphere gyri much more simple in general architecture, especially parietal and at junction of occipital and temporal regions.
XVIII (1593).	*No evidence of hallucinations.*	General atrophy or hypoplasia of cortex, with emphasis in frontal and central regions.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
XIX (1602).	Auditory hallucinations (or ideas of reference?) at first; later, paranoic picture.	Chronic internal hydrocephalus; chronic leptomeningitis of vertex; no correlations with auditory hallucinations, if such existed.
XX (1603).	*No evidence of hallucinations.*	Anomaly of superior temporal gyrus, like that of Cases II and III (*clinical correlation absent*).
XXI (1615).	Auditory hallucinosis (perhaps elaborate *Gedankenlautwerden*).	Opercular atrophy, left.
XXII (1616).	Auditory hallucinations? (God's commands).	Superior temporal anomaly.
XXIII (1622).	Auditory hallucinations fairly constant.	No gross temporal-lobe correlation (the microscope shows considerable change).
XXIV (1625).	*No evidence of hallucinations.*	Temporal lobes simple in architecture, but no evidence of atrophy.
XXV (1634).	*No evidence of hallucinations* (visual?).	Grossly, negative.

There are 12 cases in the 25 which are recorded to have had hallucinations, although in a few instances it is possible that ideas of reference or other mental states have been mistaken for hallucinations. If we take the usual clinical ground that there are more hallucinations than the examiner is ever likely to prove, it is perhaps safer at first to count these all as hallucinations; our anatomical correlations could only show a lowered index if we adopt this course.

Of the 12, there are three cases which yield no convincing gross anatomical correlation.

Case VI is described as having had one episode of auditory hallucinations. This patient died in his 17th year (he was very possibly a high-grade imbecile), and the episode in question occurred 16 months before death.

Case XIX was an almost purely delusional case, but apparently had one episode of hearing imaginary enemies talking. There was no evidence in the gross anatomy of the brain of a special location of lesions in the temporal lobe. (Microscopically there

were mild lesions in many areas, but not marked in the temporal areas; in fact, microscopically the most severe lesions were those of the two postcentral gyri, possibly related with the delusions of " dynamo shocks," " heavy feelings in limbs," etc.)

Case XXIII is perhaps the best exception to the rule of temporal-lobe correlations with auditory hallucinosis, since the two cases just mentioned had mere brief episodes of hallucinosis, whereas this case must be counted as having numerous periods of frank hallucinosis through a period of 10 years. I am here forced to fall back on the fact that microscopic examination yielded considerable evidence of cell loss in the superior temporal gyri (although less than elsewhere, and especially less than in the parietal regions—the case was in the main a catatonic one).

Accordingly I conclude that the great majority of hallucinating cases of dementia præcox exhibit at autopsy and upon careful gross brain analysis rather striking evidences of temporal-lobe involvement. Exceptions are cases with single brief episodes of hallucinosis. The one apparently genuine exception (a paranoid and catatonic case, given to characteristic long periods of auditory hallucinosis) did show moderately severe microscopic lesions; but for unknown reasons the configuration or size of the temporal lobe had not been modified.

But, even if the validity of this correlation be granted, it may well be asked whether there are not numerous other cases which show marked temporal-lobe changes *without* auditory hallucinosis. On *à priori* grounds one must naturally answer this question affirmatively, and for a much wider field of cases than the dementia præcox group. Surely the conditions which favor the hallucinating process must be quite special, and it is highly improbable that any random injury to the temporal lobe, whether wrought in or after the period of development, should in and of itself start up auditory hallucinations. I therefore expected to find fairly numerous examples of temporal-lobe change without auditory hallucinosis, all the more because the histories and records of our patients are not always held to the highest level of accuracy.

The cases that illustrate non-correlation of gross anatomical changes found with hallucinosis number at most four:

Case IV, a hyperphantasia case, might well have shown auditory hallucinations, as well as what he did show, viz., delusions

and catatonic phenomena. Some of the hyperphantasia expressed by this patient was of a sort possibly to be ascribed on theoretical grounds to temporal-lobe involvement or to a simultaneous involvement of both parietal and temporal lobes.

Case X showed temporal lobes of unusually simple architecture, and they appeared to be subject to a moderate degree of atrophy. The only suggestive correlation so far obtained is mannerisms of speech, which I suppose it is conceivable to attribute in certain instances to some disorder of word or phrase storage. But the history of this case has unfortunately not yet been properly followed out.

Case XX, another hyperphantasia case, was one of a special group showing anomalies of the superior temporal gyri. There are indications in the history that this patient may have had auditory hallucinations, although I have left them out in the above table.

Case XXIV showed temporal lobes of unusually simple architecture, but no gross atrophy had been developed.

To sum up concerning the lack of expected hallucinosis: Four of 13 cases *without* auditory hallucinosis showed temporal-lobe changes, (1) a case with unduly simple temporal lobes without atrophy, (2) a case similar to (1) but with a moderate atrophy, (3) a case with general hypoplasia (and atrophy?) in which infra-Sylvian tissues especially shared, and (4) a case with superior temporal anomaly similar to others in the series (all the others showed hallucinosis, and this case may have). The latter two cases, mentioned in the previous sentence (viz., Cases IV and XX), were cases of hyperphantasia, in which it is possible that temporal-lobe functions may play a part.

On the whole, the small percentage (three or four in 13) of cases having temporal-lobe changes without hallucinosis is, I think, smaller than one would *à priori* predict. One may argue therefrom that in dementia præcox patients temporal-lobe changes surprisingly often take such a form as to permit auditory hallucinosis.

Harking back to the correlation just preceding, it is clear that one may reason even more safely from the fact of hallucinosis to the probability of temporal-lobe disorder than from the existence of temporal-lobe disorder to the history of hallucinosis.

The point probably lodges in the fact that quite special conditions after all determine the occurrence of hallucinations, and that not every temporal-lobe disorder will serve. One must remember also that we are dealing with dementia præcox, and with dementia præcox only, in which disease possibly special stratigraphical disease occurs in which special cells alone are altered or destroyed.

With the two small groups of exceptions just mentioned, the general correlation of auditory hallucinosis with temporal-lobe disorder may be considered to be provisionally established, at least for the dementia præcox group.

CORRELATIONS OF PARIETAL-LOBE LESIONS WITH CATATONIA.

Following is a table designed to bring out the evidence for or against my formerly suggested correlation between catatonia and post-Rolandic (postcentral and other parietal) lesions. I have included all cases both catatonic and not catatonic; and in the right-hand column I have placed all evidences of parietal involvement, whether the cases were or were not catatonic.

TABLE XIV.

Gross Anatomical Correlations with Catatonia.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
I (1297).	*No certain evidence of catatonia* (outbursts of profane or obscene speech, quarrelsomeness, incoherent letters).	Left hemisphere above Sylvius and forwards of parieto-occipital fissure atrophic, but maximal atrophy is not parietal; post-Rolandic tissues of right side more atrophic than left.
II (1317).	Catatonia late in course (resistivism, mutism, refusal to eat, violence; later, apathy, manneristic speech, grimaces).	The only gross lesion behind Rolando was a tendency to occipital microgyria; *parietal correlations absent.*
III (1319).	Catatonia? (resistive but fearful, tube-fed at times).	No special parietal-lobe change (unless affected by the chronic internal hydrocephalus, more marked on left).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
IV (1335).	Catatonia gradually supplanted paranoic symptoms (catatonic excitements, mannerisms, fixed positions, inability to walk a straight line).	Parietal emphasis of the general brain atrophy (possibly hypoplastic at onset).
V (1383).	Catatonia (mannerisms, spasmodic movements of jaw, lid-closure).	Postcentral atrophy, more marked on right. Internal hydrocephalus, especially of mid portions of ventricles.
VI (1413).	Episode probably catatonic.	Callosomarginal anomalous folding above splenium on both sides.
VII (1472).	Nothing catatonic until development of peculiar stepping movements toward close of long course.	General slight brain atrophy.
VIII (1487).	Catatonia (three weeks' course).	No sclerosis or atrophy anywhere; asymmetry of brain.
IX (1491).	Catatonia (head constantly uplifted in bed, mutism, *cerea flexibilitas*, resistivism, negativism).	Chronic internal hydrocephalus; mild atrophy (aplasia?) of paracentral lobules.
X (1507).	Mannerisms of speech.	No parietal-lobe lesions.
XI (1509).	Catatonia (excitements, impulsive acts, mutism, resistivism, refusal of food, mannerisms, limp muscles).	Moderate chronic internal hydrocephalus, moderate parietal atrophy.
XII (1518).	Catatonia (periods of excitement, negativism, resistivism).	Callosomarginal acquired lesion, right; developmental disturbance of same region, left; chronic internal hydrocephalus, more marked posteriorly.
XIII (1540).	Catatonic resistivism, mannerisms of posture, impulsive violence.	General mild atrophy and sclerosis of brain; maximal atrophy (or aplasia?) of left angular gyrus; chronic internal hydrocephalus, more marked posteriorly.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
XIV (1571).	*No evidence of catatonia* (somatic delusions).	No parietal-lobe change.
XV (1574).	No evidence of catatonia.	No parietal-lobe changes.
XVI (1575).	Some symptoms are allied to catatonia (peculiar "tranquil" violent kicking of patients, etc.).	General brain atrophy (especially superior and inferior parietal regions).
XVII (1583).	Catatonia (*cerea flexibilitas,* impulsive violence, resistivism).	Left-hemisphere gyri much more simple in general architecture than right, especially parietal and at junction of occipital and temporal gyri; moderate general brain atrophy; asymmetry.
XVIII (1593).	Catatonia (catalepsy, *cerea flexibilitas,* neologisms, mutism).	Postcentral atrophy, well marked, along with precentral and superior frontal atrophy.
XIX (1602).	*No evidence of catatonia.*	Marked chronic internal hydrocephalus; chronic leptomeningitis of vertex.
XX (1603).	*No evidence of catatonia.*	Marked chronic internal hydrocephalus; no special parietal involvement.
XXI (1615).	*No evidence of catatonia* (one "hysterical" attack).	Inferior parietal atrophy.
XXII (1616).	Catatonia (mannerisms, violence).	Chronic internal hydrocephalus (no special parietal involvement).
XXIII (1622).	Catatonia late in course (mutism, resistivism).	Superior parietal atrophy; left supramarginal atrophy most marked.
XXIV (1625).	Catatonia (resistivism, negativism, verbigeration, fixed attitudes, *cerea flexibilitas*).	Chronic internal hydrocephalus confined to posterior halves of bodies of lateral ventricles; thinning of posterior part of corpus callosum; atrophy of inner surfaces of occipital lobes.
XXV (1634).	Catatonia (*cerea flexibilitas,* mannerisms, impulsive movements).	Grossly, negative.

My clinical analysis yields 14 cases in which there seems to be no doubt that catatonic symptoms definitely appeared, and there are seven others in which catatoniform or allied symptoms are recorded. Four were quite negative.

Of the 14 definitely catatonic cases (and here I do *not* mean cases which would necessarily be placed in Kraepelin's catatonic *form* of dementia præcox, either in his formulation of 1899 or in that of 1913; but I refer to cases having *symptoms* of this general nature), 10 at once yielded parietal or other correlations behind the fissure of Rolando. Two of four others were the two grossly negative cases of brief duration (VIII and XXV) in which no gross *acquired* lesions of an atrophic nature could have been expected (*they did show microscopic changes,* as elsewhere mentioned).

This analysis leaves outstanding two cases with absence of gross correlations. II developed catatonia late, and the only gross lesion or anomaly behind Rolando was a tendency to microgyria of the occipital regions. XXII showed mannerisms and violence supposed to be catatonia; and the brain showed chronic internal hydrocephalus, but without emphasis upon any region behind Rolando.

If we count II and XXII as without correlation (although this is hardly a fair account of either), still the percentage of incidence of posterior-lying lesions in catatonia stands at 85 per cent.

Of the seven cases in which our interpretation must remain doubtful as to whether the symptoms were catatonic or not, but which may well have been catatonic, I find five with various posterior-lying lesions, and two without such correlation.

In four cases in which we can apparently exclude catatonic symptoms entirely, there were no posterior-lying lesions.

Analyzing from the standpoint of catatonia, accordingly, the parallelism is very striking between catatonia and lesions of the cortical arrival-platforms and the posterior association center. But, as in the case of hallucinosis and the temporal region, it is only fair to correlate in the other direction, from lesion to symptom. I will not here repeat what was said above under hallucinosis, to the effect that correlations are likely to be less strong when running from lesion to symptom than from symptom to lesion.

We seem to be able to exclude all gross lesions and anomalies of whatever kind (so far as posterior-lying lesions go) in Cases VIII, X, XIV, XV, XXV, of which the first and last (VIII and XXV) are the grossly negative cases in which we must rely on microscopic changes, and of which X showed merely mannerisms of speech, and XIV and XV showed no catatonia.

Thus there were 20 cases in which the brain showed either a special emphasis of posterior-lying lesions in the gross or involvement of these areas in a general or universal brain change.

Taking these in order we find all clearly catatonic except the following:

I. (Outbursts of profane or obscene speech, quarrelsomeness, incoherent letters.)
III. (Catatonia?—resistive but fearful, tube-fed at times.)
VI. (Episode probably catatonic.)
XVI. (Symptoms allied to catatonia.)
XIX. (No evidence of catatonia.)
XX. (No evidence of catatonia.)
XXI. (One "hysterical," attack.)

Thus 65 per cent of the cases showing either general or special involvement of the posterior-lying areas were catatonic, and some suspicion attaches to 25 per cent more, leaving 10 per cent with absolutely negative correlations. These two cases, XIX and XX, were cases of marked internal hydrocephalus, but without special emphasis.

If these figures can be taken on their face-value, it may well be supposed that not every case which exhibits posterior involvement will necessarily produce catatonic symptoms to correspond and that very possibly special conditions (cytological, stratigraphical, microphysical) have to do with the appearance or non-appearance of these symptoms. In particular, however often catatonia is correlated with internal hydrocephalus, it does not yet appear that the conditions producing the hydrocephalus necessarily produce catatonia, although for unknown reasons they seem more likely to produce catatonia (as well as hallucinosis) than to produce delusions.

Special attention should be given to *cerea flexibilitas* as a catatonic symptom *par excellence*. Following are cases:

IX. (Internal hydrocephalus; slight atrophy (aplasia?) of paracentral lobules.)
XVII. (Moderate general brain atrophy, with apparently greater involvement of parietal region and zone of junction of temporal and occipital regions on left side (due to aplasia?).)
XVIII. (Postcentral atrophy, well marked.)
XXIV. (Internal hydrocephalus limited to posterior halves of bodies of lateral ventricles; thinning of posterior part of corpus callosum; atrophy of inner surfaces of occipital lobes.)
XXV. (Grossly, negative; microscopically, positive case.)

In this series of cases with *cerea flexibilitas,* the tendency to parietal involvement is especially plain, and inasmuch as two of the four cases with lesions showed the gross emphasis of these lesions in or near the sensory arrival-platforms for touch and muscle-sense, it may well be supposed that certain conditions in those areas underlie *cerea flexibilitas.* From the nature of that symptom itself, it may be surmised that its relation to disordered kinæsthesia may some day be established.

CORRELATIONS OF FRONTAL-LOBE LESIONS WITH DELUSIONS.

Following is a table designed to bring out the truth or falsity of the *paranoia-frontal-lobe* correlation for which I endeavored to lay a foundation in the 1910 paper (see conclusions 12 and 14 from that paper, quoted above).

It should be said that I use the term paranoia in the sense of a symptom, *not* in the sense of an entity. Moreover, I use it in a broad sense to indicate " insane delusions " of all sorts, not in the narrower sense of " delusions of persecution." The correlation we here investigate is accordingly between the delusional character and brain conditions, between the acquired or dispositional tendency to false beliefs (or better, the habit of falsely believing) and disease process in whatever we may discover to be the believing-mechanism in the brain.

In parentheses in the left-hand column are placed also the data concerning hallucinosis, transferred in brief from the previous table.

TABLE XV.

GROSS ANATOMICAL CORRELATIONS WITH PARANOIA (INSANE DELUSIONS).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
I (1297).	Delusions, allopsychic (with hallucinosis).	Entire *left* hemisphere above Sylvius and forwards of parieto-occipital fissure atrophic; maximal atrophy about right first frontal sulcus; corpus callosum *not* thinned.
II (1317).	Delusions, religious, and fear, probably both autopsychic and allopsychic (with hallucinosis).	Left superior frontal gyrus smaller than right (developmental disorder with superadded atrophy?).
III (1319).	Delusions, religious (regarded as based on hallucinosis).	No special frontal-lobe correlate (unless affected by the chronic internal hydrocephalus, more marked on left).
IV (1335).	Delusions, allopsychic, fantastic, supernatural agency (without hallucinosis).	General atrophy of brain (possibly hypoplastic at outset), but with parietal and temporal emphasis.
V (1383).	No evidence of delusions (long-standing case with little early history).	General brain atrophy, more marked on right, and more marked forwards of Rolando. Internal hydrocephalus, especially mid portion.
VI (1413).	Delusions, allopsychic, perhaps dominantly autopsychic; change of character, possibly high-grade imbecile (one episode of hallucinosis).	Tendency to frontal atrophy, more marked on left.
VII (1472).	Delusions, allopsychic, persecutory, with tendency to hyperphantasia (with questionable hallucinosis).	General hypoplasia, or more probably atrophy, of brain; second and third frontal gyri tend to show greater atrophy.
VIII (1487).	Delusions, religious, self-accusatory, at first; later characteristically catatonic.	No gross lesions; asymmetry of brain (three weeks' duration).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
IX (1491).	History inadequate; a few delusions probable, allopsychic (no history of hallucinosis).	No frontal-lobe lesions, except tendency to mesial thinning (but this was more marked about paracentral lobule); chronic internal hydrocephalus.
X (1507).	Delusions, allopsychic.	Frontal atrophy or aplasia of milder degree than the temporal-lobe atrophy or aplasia (which was itself moderate).
XI (1509).	Delusions may possibly be inferred from hyper-religiosity (hallucinosis inferred from attitudes).	Moderate frontal atrophy (along with central and parietal atrophy); moderate chronic internal hydrocephalus.
XII (1518).	Delusions—allopsychic?—inferred from certain replies (without hallucinosis).	Left-sided frontal aplasia; slight chronic internal hydrocephalus.
XIII (1540).	Delusions, sexual, as well as others inferred from evasive and reticent attitude (hallucinosis surmised).	Mild atrophy (or aplasia?) of both superior frontal regions (mesial as well as lateral).
XIV (1571).	Fantastic somatic delusions, probably on a peripheral basis (without hallucinosis).	No special frontal involvement; but marked asymmetry (distortion?) of brain.
XV (1574).	Delusions, autopsychic, often fantastic in content (without hallucinosis).	Frontal (as well as parietal) opercular atrophy.
XVI (1575).	Fantastic delusions, allopsychic—and autopsychic? (without hallucinosis).	General cortical atrophy, especially superior and inferior parietal.
XVII (1583).	Delusions, allopsychic, somatic (with hallucinosis).	No special frontal-lobe involvement (except that left is simpler than right; this disparity is still greater in parietal and at junction of occipital and temporal regions).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
XVIII (1593).	Delusions, sexual, later grandiose (without hallucinosis).	General cortical atrophy, especially superior frontal and central (corpus callosum not thinned).
XIX (1602).	Fantastic delusions, allopsychic, autopsychic, somatic (hallucinosis questionable).	No special frontal involvement (except for marked chronic internal hydrocephalus); thinning of posterior part of corpus callosum.
XX (1603).	Delusions, allopsychic, perhaps dominantly autopsychic; hyperphantasia (without hallucinosis).	No special frontal involvement (except for marked chronic internal hydrocephalus); marked chronic leptomeningitis over left frontal operculum.
XXI (1615).	Delusions—ideas of reference; perhaps elaborate *Gedankenlautwerden* (question of hallucinosis).	No frontal-lobe involvement, except opercular atrophy, more marked on left; inferior parietal atrophy.
XXII (1616).	Delusions, autopsychic, allopsychic? (hallucinosis doubtful).	No frontal-lobe involvement (except possible effects of chronic internal hydrocephalus).
XXIII (1622).	Delusions, autopsychic, allopsychic (with hallucinosis).	No frontal correlation.
XXIV (1625).	Delusions questionable—ideas of reference (without hallucinosis).	Inferior frontal gyri aplastic? or atrophic?
XXV (1634).	Delusions, allopsychic (without hallucinosis).	Grossly, negative.

There is an *embarras de richesse* in the business of anatomical correlations with delusion-formation. Both delusion-formation and frontal-lobe atrophy are common phenomena, and perhaps we hesitate to admit their correlation (to the end of establishing any sort of causal relation) just by reason of the frequency of their appearance.

There is but one case in the series in which there is no evidence of delusion-formation; that case (V) exhibited a generalized brain atrophy, more marked on the right side and *forwards* of the

fissure of Rolando, as well as internal hydrocephalus, especially of the mid portions of the lateral ventricles. The history, duration of disease, and the very age of the patient are all unknown to us, although (as the case-description shows) there can be little question of the appropriateness of the diagnosis dementia præcox from the terminal symptom picture. I should hesitate to break the force of any correlations concerning delusion-formation on the evidence of this case. Note is made of a certain circumstantial loquacity occasionally breaking forth and of various peculiar ways of thinking, which may represent residuals of a frontal-lobe disorder early more marked.

Of the 24 cases with delusion-formation more or less in evidence, seven exhibited no gross frontal-lobe disorder. Two of these are the two entirely negative cases (from the gross point of view), viz., VIII and XXV. Both these cases, as elsewhere mentioned, yielded ample microscopic evidence of frontal-lobe disease.

The five remaining cases of delusion-formation without frontal-lobe correlations deserve special comment.

XIV was a case presenting well-marked fantastic delusions of a somatic nature (which I consider probably had a peripheral basis). Like several others of the " hyperphantastic " group, this case exhibited no special frontal involvement, but, unlike several, failed to show special parietal involvement (see separate discussion of the hyperphantasia group, as also remarks in the case-description above). I am inclined to believe, with my colleague, Dr. H. M. Adler, that the future may show that the peculiar delusions of bodily distortion may be somehow correlated with acquired or progressive skull-distortion. Such is the peripheral basis which I mentioned above; but when the skull itself is the seat of changes, it is a question of physics which parts of the brain will respond symptomatically. I prefer to align this case with several reported in my communication " On the Somatic Sources of Somatic Delusions."

XVII was another case of somewhat less fantastic delusions of a similar somatic nature. The only delusions of an apparently somatic nature were some of poisoning, which may well have been founded on hallucinosis (" poison in saliva "). As I read the case, possibly all the delusions were founded on hallucinosis, or

actual morbid somatic phenomena, or on a fanciful rendering of normal somatic phenomena. Anatomically, the left frontal region was of simpler construction than the right, but there was no evidence of frontal-lobe atrophy.

XXI was also a case of unusual nature, and the attitude was possibly largely one of elaborate *Gedankenlautwerden* (long archaic prayers in French, etc.) in which there may have been a strong hallucinational nucleus. There was in this case no special frontal involvement, except that the left inferior frontal region was atrophic.

XXII yielded autopsychic and possibly allopsychic delusions, but showed no special frontal-lobe disorder, unless we may suppose effects wrought by internal hydrocephalus. The delusions mentioned are somewhat doubtful, and are perhaps in part due to auditory hallucinosis (" heard God's commands ") ; and the case early turned into a strongly catatonic case.

XXIII was a case of pronounced autopsychic delusions, combined with (or largely based upon?) somatic delusions. The hallucinational and catatonic symptoms were equally marked. Anatomically, the case falls more in the parietal group. Microscopically, there was considerable evidence of frontal involvement, but again the emphasis was parietal.

No one of these cases is strong enough to break the general correlation; indeed, three of the cases of apparent exception to the frontal-delusional correlation, viz., XIV, XVII, and XXI, go far to illustrate the concept of what might be termed sensorial delusions, considered elsewhere (hyperphantasia group).

CASES WITH FANTASTIC DELUSIONS (HYPERPHANTASIA).

I was led to distinguish, above, the large group of cases of ordinary delusion-formation from a small group in which a varied succession of absurd and fantastic delusions was developed. Upon theoretical grounds, holding to the correlation of ordinary delusion-formation with lesions of the anterior association-center, I was inclined to consider that the basis of the hyperphantasia might lie in the posterior association-center. Let us now consider this question on its merits.

HYPERPHANTASIA.

IV. Lesions (aplasia and atrophy) universal, but more marked in occipital and temporal regions.

VII. Lesions (aplasia and atrophy) universal, but more marked in temporal and middle frontal regions.

XVI. Lesions (atrophy) universal, but more marked in regions behind Rolando and above Sylvius, and most marked in superior and inferior parietal regions.

XX. Internal hydrocephalus and left-sided superior temporal anomaly.

XXI. Internal hydrocephalus (doubtful) and parietal atrophy.

In the above five cases the delusions were such as to attribute fantastic features to the environment. In two others the fantastic features were attributed to various parts of the bodies of the patients.

XIV. Correlation proposed with (acquired?) asymmetry of skull.

XIX. Posterior-column degeneration of spinal cord; internal hydrocephalus; chronic leptomeningitis.

In the entire group of seven cases it will be observed that universal lesions or conditions of unusual character prevail, as a rule either generalized atrophy or internal hydrocephalus (though not both). Accordingly, no area can be safely excluded from a share in the effects. Hydrocephalus in any event seems to affect the posterior-lying portions of the brain more than the anterior-lying portions. Where gross atrophy appears, it is exhibited as a rule most markedly in the parietotemporal regions.

Concerning the somatic hyperphantasia cases, one should not dogmatize, although I seem to find adequate somatic or sensorial nuclei for the delusions developed by these cases.

For the non-somatic group of hyperphantasia cases, I am inclined to vindicate the parietotemporal correlation, not omitting to concede, however, that the frontal regions are also as a rule to some extent involved.

VI. CONCLUSIONS.

1. The writer has followed up his earlier work on the dementia præcox group (1910) with a more systematic anatomoclinical study of 25 cases, having a view to (a) definite conclusions as to the structurality ("organic nature") of the disease, and (b) correlation of certain major symptom groups (delusions, catatonic symptom groups, auditory hallucinosis) with disease of particular brain regions.

2. As to (a), the *structurality of dementia præcox,* the writer feels that the disease must be conceded to be in some sense structural, since at least 90 per cent of all cases examined (50 cases, data of 1910 and 1914) give evidence of general or focal brain atrophy or aplasia when examined post mortem, even *without* the use of the microscope.

3. Moreover, *with* the use of the microscope, the problem of the normal-looking remainder can perhaps be solved, since the only two normal-looking brains[7] in the 1914 series of 25 yielded abundant appearances of cell-destruction and satellitosis in the cerebral cortex, which had not yet had time to be registered in the gross (cases of three weeks' and two months' duration respectively).

4. The method of anatomical analysis in the new series is a more systematic one than has been hitherto employed, involving careful gross description of the fresh brain; careful preservation (by suspension from basal vessels) in formaldehyde solution; systematic photography to scale of the superior, inferior (cerebellum removed), lateral, and mesial aspects before and after stripping the pia mater; study of all aspects of the brain as spread side by side in photographic form; further study of the preserved brains in the light of the photographic study; and eventual cytological or fiber studies of paired structures showing possible atrophy or aplasia.

5. The neuropathologist making such a brain analysis shortly discovers that there is often more to be learned from the gross than from the microscopic appearances, since, of two gyri, the one measurably smaller than the other (and therefore probably agenesic, aplastic or atrophic), the microscopic appearances may often be hard to diagnosticate, as the normal-looking gyrus at the time of death may be just undergoing a satellitosis actually indicating more disease than its shrunken fellow.

6. Nevertheless, the gross analysis gives one perfectly convincing evidence of some kind of lesions, leaving to other methods of study the decision as to the congenital or acquired nature of these lesions. Some 14 of the 25 cases may be regarded as in some sense maldevelopmental, so as to arouse the suspicion that the acquired atrophy was grafted on top of a congenital agenesia or aplasia; but, in the opinion of the writer, aplasia is indicated rather than

agenesia: the potential victim of dementia præcox is probably born with the normal stock of brain cells, although their arrangement and development are at times early interfered with.

7. The atrophies and aplasias, when focal, show a tendency to occur in the left cerebral hemisphere. The coarse atrophy is usually of only moderate degree, and often does not appreciably alter the brain weight, at least outside the limits of expected variation. In fact, the heart, the liver, the kidneys, and the spleen tend to show greater loss in weight than does the brain.

8. More remarkable than the atrophy and aplasia of the cortex is the high proportion of cases of internal hydrocephalus (at least nine cases) uncovered by the systematic photographic study of frontal sections.

9. There is no evidence that this internal hydrocephalus is due to generalized brain atrophy. It is possible that it begins more posteriorly. It is probable that it does not mechanically so much affect the frontal lobes. It is associated with cases of long duration, although not with all cases of long duration, and was never found in cases of brief duration. Clinically, the hydrocephalic cases are uncommonly catatonic, and the cases of marked generalized hydrocephalus were as a rule victims of hallucinations. Delusions, except fantastic delusions, were not prominent in this group. The clinical courses of these hydrocephalic cases were more than usually active and mutable, and were often interrupted by remissions.

10. The hydrocephalic brains were not in other respects particularly open to the suspicion of congenital disease; and, without adequate proofs, the writer is inclined to consider the hydrocephalus to be often an acquired hydrocephalus.

11. An ardent supporter of congenital features might claim that 19 of the 25 brains showed some sort of maldevelopmental defect; one impartial witness thought that 14 showed such; and even if all nine cases of hydrocephalus be taken as acquired, we remain with 11 cases bearing pretty certain evidence of maldevelopmental defect. On the other hand, all but six cases showed signs of acquired lesion, and these six showed various microscopic changes of doubtful meaning, but certainly acquired.

12. One remains with the general impression that gross alterations are almost constant and microscopic changes absolutely con-

stant, and that the high proportion of gross appearances suggesting aplasia means that structural (visible or invisible) changes of a maldevelopmental nature lie at the bottom of the disease process. But this suspicion of underlying maldevelopment is only a suspicion, although a strong one, and the first factor for the theory of pathogenesis to explain is the gross and microscopic changes as they present themselves in the full-fledged case.

13. Aside from left-sidedness of lesions and internal hydrocephalus, very striking is the preference of these changes to occupy the association-centers of Flechsig. For this there is probably good *à priori* reason in the structure, late evolutionary development, and consequent relatively high lability of these regions. The interest of these findings is still greater in the functional connection (see below).

14. In concluding this summary of the anatomical side of the study, the writer cannot forbear adding that he supposes many neurologists, hearing of "lesions," will at once imagine extirpatory lesions of a Swiss-cheese appearance or areas like those of tuberous sclerosis. At the risk of being charged with *naïveté,* the writer would again here insist that the lesions described, though never beyond the range of a skilful anatomist, are of a mild atrophic nature or in the nature of aplasias, requiring care and deliberation in their description and explanation, and often hard to grasp except where photographs of all sides of the brain may be compared at once and reference then made to the brains themselves. These lesions do not effect globar lacunæ in the cortical neuronic systems, but they are of a more finely selective character. Under the microscope it may be difficult to say, without elaborate micrometry, that one area is worse off than another; but convincing evidence of the gross convolutional extent of the process is got by the naked eye and by the finger.

15. The writer regards this work as putting the burden of proof on those who claim the essential functionality of dementia præcox, and is at some pains to couch objections to one formulation of these changes as "incidental," and to another, as "agenesic." Nevertheless, the writer would not necessarily deny the value of those formulations which look on these cases as cases of faulty adaptation to environment.

16. As to (b), the *functional correlations* of this study, the results may be summed up by saying that strong correlations have been found to support the writer's former claims that (1) delusions are as a rule based on frontal disease, and (2) catatonic symptoms on parietal-lobe disease. An equally strong correlation (3) has now been found between auditory hallucinosis and temporal-lobe disease.

17. The writer's previous work had suggested a correlation beteween frontal-lobe disease and delusion-formation. This correlation is not so decided in the present series, since, although perhaps only one of the 25 cases failed to exhibit delusions, seven of the remaining 24 failed to show frontal-lobe lesions. However, two of these seven, though grossly negative, were microscopically positive enough.

18. The findings indicate, accordingly, that there is a group of delusional cases such that even long duration does not determine a frontal emphasis of lesions. Five cases represent this exceptional condition: three of these five are probably best interpreted as cases of hyperphantasia in which, both *à priori* and by observation, frontal lesions are not characteristic.

19. On the whole, the correlation between delusions and focal brain atrophy (or aplasia capped by atrophy?) is very strong, particularly if we distinguish (1) the more frequent form of delusions with frontal-lobe correlations from (2) a less frequent form with parietal-lobe correlations.

20. The non-frontal group of delusion-formations, the writer wishes to group provisionally under the term *hyperphantasia,* emphasizing the overimagination or perverted imagination of these cases, the frequent lack of any appropriate conduct-disorder in the patients harboring such delusions, and the *à priori* likelihood that these cases should turn out to have posterior-association-center disease rather than disease of the anterior association-center. This anatomical correlation is in fact the one observed.

21. The writer's previous work had suggested a possible correlation between catatonic phenomena and parietal (including post-central) disease: 10 of 14 definitely catatonic cases yielded parietal or other post-Rolandic lesions; two were grossly negative but microscopically altered; and indications of correlation appeared also in the remaining two. Five of seven clinically some-

what doubtfully catatonic cases yielded similar correlations. Four clinically non-catatonic cases yielded no parietal correlations. (It is worth while insisting that "catatonia" is here used to refer to a symptom, *not* to an entity or clinical group.)

22. Special interest attaches to *cerea flexibilitas* as a clearly definable form of catatonic symptom: four of five cases yielded gross parietal lesions. The fifth case was one of the entirely negative cases in the gross, but showed very marked postcentral satellitosis microscopically. Two of these cases showed the gross emphasis of lesions in the postcentral gyri, thereby hinting at an explanation of *cerea flexibilitas* along the lines of a reaction to altered kinæsthesia or an altered reaction to normal kinæsthesia (depending upon such true analysis of intragyral cortex-function as the future may bestow).

23. *À priori* one might expect a correlation between the characteristic auditory hallucinosis found in many cases of dementia præcox and temporal-lobe lesions. In point of fact, nine of 12 hallucinated cases yielded temporal-lobe atrophy or aplasia; and actually only one of the three others is a good exception to the rule (from the clinical standpoint), to say nothing of the fact that this case had ample microscopic changes in the temporal lobe.

24. Of the 13 *non*-hallucinating (auditory) cases, only three, or at most four, could be said to have temporal-lobe lesions suggesting the possibility of hallucinosis; here we may appeal to the inadequacy of clinical work, or, better, to the non-suitability of the lesions, since no one would assert that we yet have any idea of the precise and intimate temporal-lobe conditions which permit hallucinations.

25. In these functional connections, the more recent formulations of Kraepelin and of Bleuler have been reviewed, although the entire work was done without the benefit of their analyses. The present formulation appears consistent enough with either. It would seem that Kraepelin regards a correlation between auditory hallucinosis and temporal-lobe disease as already highly probable from the literature. He also goes so far as to incriminate the "central" region for motor disorders. But the present suggestions as to the possible kinæsthetic relations of catatonia and the special (frontal and parietal) correlations with delusion-

formation are not suggested by Kraepelin from the literature available.

26. It is interesting to note that further study by the Munich workers seems to have drawn attention away from the *infrastellate* cortical changes sketched by Alzheimer for catatonia in 1897 to various *suprastellate* changes. The microscopic work done in the present study in connection with certain grossly negative cases indicates that the early phases of the process may very often look as if infrastellate change was to be the most striking product of the disease. This is perhaps due to a richer original supply of glia cells in these infrastellate layers. Later, when the process is less acute, it may often be found that suprastellate cell losses are much more in evidence than any striking infrastellate change.

27. As for the general position which this work would assume toward the functional conclusions of Bleuler, it would seem that a histopathological basis for " dissociations " or " schizophrenia " could be somewhat readily provided by the lesions found, since these are for long periods mild enough and sufficiently confined to the finer cortical apparatus to provide for the exquisite mental changes of most cases. The main neuronic systems are often permanently preserved, leaving an irregularly and slightly simplified cortical apparatus, in which a few cell changes would naturally throw out of coordination a great deal of still intact apparatus. But the whole process often remains so mild as to permit reestablishment of relatively normal functional relations on a slightly simplified basis, the whole to be disturbed once more on the occasion of the death or disease of a few more cells. Very striking is the fact that the cells not attacked are, so far as we can see, normal enough.

28. This work is rather a study of genesis than of etiology, in the sense of modern medical distinctions between these branches of inquiry. It is a modest inquiry into factors, and does not rise to the height of ascribing causes. The writer will refer merely to some paragraphs in the text as to a possible ontological position concerning structure and function which the future may take. The deplorable thing is that some structuralists throw out of court all functional data and some (rather more!) functionalists tend to underrate the possible contributions of anatomy to this field. Luckily, science nowadays cannot long proceed merely *à la mode*.

29. In particular, to sum up, I would call especial attention to the following points: (1) The constancy of mild general or focal atrophies in cases lasting long enough to yield these; (2) the tendency to an exhibition of lesions somewhat more markedly in the left hemisphere; (3) the preference of the lesions for the "association-centers" of Flechsig; (4) the high correlation of auditory hallucinosis and temporal-lobe lesions, as also (5) of catatonia and parietal lesions (*cerea flexibilitas,* especially postcentral), and (6) of the more frequent form of delusions and frontal-lobe disease; (7) the possible existence of a hyperphantasia group with parietal correlations, and of (8) a large internal hydrocephalus group with catatonic and hallucinotic correlations rather than delusional. A few more points can be got from the description of the accompanying plates.

VII. Description of Plates.

A selection of photographs has been made from the complete collection to illustrate partially the points made in the text. The collection of which these photographs are examples contains now upwards of 7000, made systematically from over 500 brains representing cases of (a) mental disease (courtesy of various state institutions for the insane, especially Danvers and Boston), (b) epilepsy (courtesy of the Monson State Hospital), (c) feeble-mindedness (courtesy of Massachusetts School for the Feeble-minded, (d) criminality and other medico-legal cases (courtesy of the Suffolk District Medical Examiner), and (e) normal and miscellaneous nature (courtesy of various institutions and physicians).

The photographs have been made (1) with the pia mater *in situ,* (2) with the pia mater stripped, and (3) in frontal sections in chosen planes. The anatomical preparations have been made largely by Dr. Annie E. Taft, Custodian of the Neuropathological Collection, Harvard Medical School, serving as a special investigator under the State Board of Insanity under the direction of the writer as Pathologist to the Board.

The photography has been executed, save in a few instances, by a professional photographer, Mr. Herbert W. Taylor.

Considerable technical aid has been contributed by the Department of Neuropathology of the Harvard Medical School, largely from gifts by Miss Katherine E. Bullard and Mrs. Zoe F. Underhill. The contributions from the Underhill foundation have been made largely to secure evidence of normality of the brain in certain cases, as her gift was for the study of non-nervous factors in nervous and mental disease.

Plate I illustrates one of the most marked degrees of *generalized atrophy* found in the series. There is, however, a tendency to *focal emphasis of atrophy* in the inferior parietal region. As usual, the *left hemisphere* was more affected than the right. Death at 34 years, onset at 22, course of 12 years. Paranoidal, later catatonic (Case XXIII).

Plates II and III present a good example of the striking *contrast* often shown *between the left and right hemispheres,* in which event the *left* hemisphere almost always presents *more atrophy* than the right. It would hardly be supposed that Plates II and III represented hemispheres from the same brain. This case was one in which disorder of glands of internal secretion may be safely asserted. Death after a course of nine months terminated by diphtheria (Case XVII).

Plates IV and V present mesial views from a brain (Case IV) which weighed 985 gm. at death (age 32, duration of symptoms 14 years, with a previous attack 20 years before death). Patient was an adequate shoe-operative, and there seems to have been no suspicion of feeble-mindedness in the sense of a congenital condition. Yet the heart (145 gm.), the aorta, the liver (945 gm.), as well as the spinal cord, were smaller than normal. Possibly there was a true agenesia with numerical loss of elements. Perhaps the case was rather one of aplasia (the writer's opinion). Case VII was a similar case (brain weight 955 gm., duration of symptoms 26 years, onset at 28), in a normal-school graduate teacher.

Plates VI to X are from Case II (death at 44, duration of symptoms 21 years).

Plates VI and VII are presented to show types of anatomical analyses. Attention is attracted to the small annectant-like gyrus in the left frontal lobe through which the line passes in Plate VI. Inspection shows that the left frontal lobe is narrower than the

right. Frontal section shows (Plate VII) that this idea is not deceptive, and that there is beyond question less substance to the left superior frontal division of the frontal region than to the right. Microscopic study (not here presented) would then be required to show whether this difference is only apparent and possibly due to the spreading over of superior frontal cortex type to a locus below the first frontal sulcus. This does not appear to be the case in the present examples. But, even if it were, the fact remains that the whole left frontal region is smaller than the right, at least in its forward part (see second frontal section, in which the difference has practically disappeared). Here, then, a contention is established that there is, anatomically at least, something to explain, be it agenesia, aplasia, or atrophy. Microscopically, there is much gliosis, though it is hard or impossible to tell whether more in sections from left or from right. But that there were fewer cells to start with in the left superior frontal gyrus than in the right can perhaps not be safely asserted; the question of agenesia *versus* aplasia is left unresolved both in the gross and in the microscopic examination, but may possibly be resolved by micrometric methods (size of cells on the two sides).

Plates VIII to X (from the same case as VI and VII) show another type of anomaly, of which there were five good examples in the whole series. The anomaly consists in an apparent "burial" of the left superior temporal gyrus. Frontal section (Plate X) shows these conditions in cross-section. The section is taken through a plane in which a local dimpling of the *right* superior temporal gyrus is also shown. (See below for another example of this anomaly.) By microscopic examination of the same loci in each superior temporal gyrus, it is possible to make a *rationale* as to which part of the gyrus has suffered most. Should one, however, rely on microscopic examination alone (without reference to the gross anomaly), the discovery of more or less well-marked gliosis in both gyri (the actual finding) would obscure the true issue as to initial agenesia or aplasia. Micrometric studies are required for this latter most important issue.

Plates XI to XIII are from Case III (death at 31, duration of dementia-præcox symptoms eight years), possibly a high-grade imbecile.

Plates XI and XII show a more pronounced example of the "burial" anomaly of the left superior temporal gyrus, together with a certain amount of cortical atrophy (aplasia?, agenesia??).

Plate XIII demonstrates the great difference between the left and right superior temporal gyri. There is also a certain degree of internal hydrocephalus, which it is to be noted is more marked on the side of the temporal anomaly. The left Sylvian fossa has much wider spaces than the right.

Plates XIV to XVI are from three different cases, and illustrate hydrocephalus.

Plate XIV was from Case XIX (possibly paraphrenia phantastica of Kraepelin's 1913 formulation), with onset at 34, duration of 28 years, death at 62. The brain was probably over weight at death (1315 gm., body length 157 cm.).

Plate XV was from Case XX, in which death was due to a massive cerebral hemorrhage. But (1) hydrocephalus and (2) superior temporal anomaly can still be made out. Death at 67, onset at 33 or younger.

Plate XVI was from Case XXII (onset at 26, death at 59). There was a small cerebral hemorrhage in this case also; possibly also a trace of the left-sided superior temporal anomaly shown in other cases.

Plates XVII to XIX are from one case (XII), with onset at 16 and death at 45. This case is being subjected to more intensive study, with the object of resolving the problem which the brain presents. Plate XVII gives the basal view, and shows what may be termed *cruciate asymmetry*. The *left* frontal region (at the right in the plate) is obviously smaller than the *right* (this is confirmed also by frontal section); but *the right hippocampal gyrus* (to the left in the plate) *is obviously smaller than the left*. Plates XVIII and XIX present mesial views of the same brain, in which the hippocampal disparity stands out well.

Can this be an example of agenesia or aplasia in which the disturbing factor acted prior to or during the decussating phase of the nervous system? That such may be the case is indicated by the reflection that most of the connections with the frontal region are very probably indirect or crossed connections, whereas the hippocampal connections are perhaps largely uncrossed connections.

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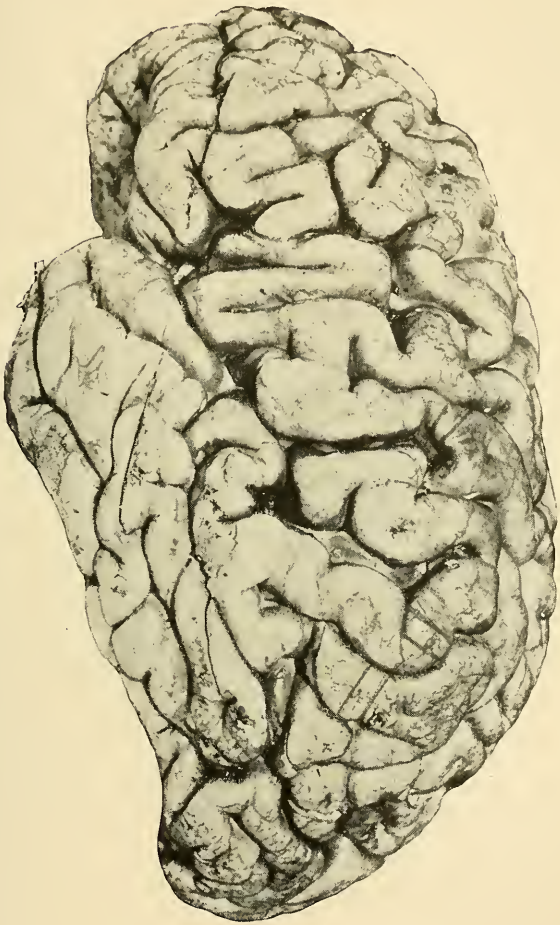

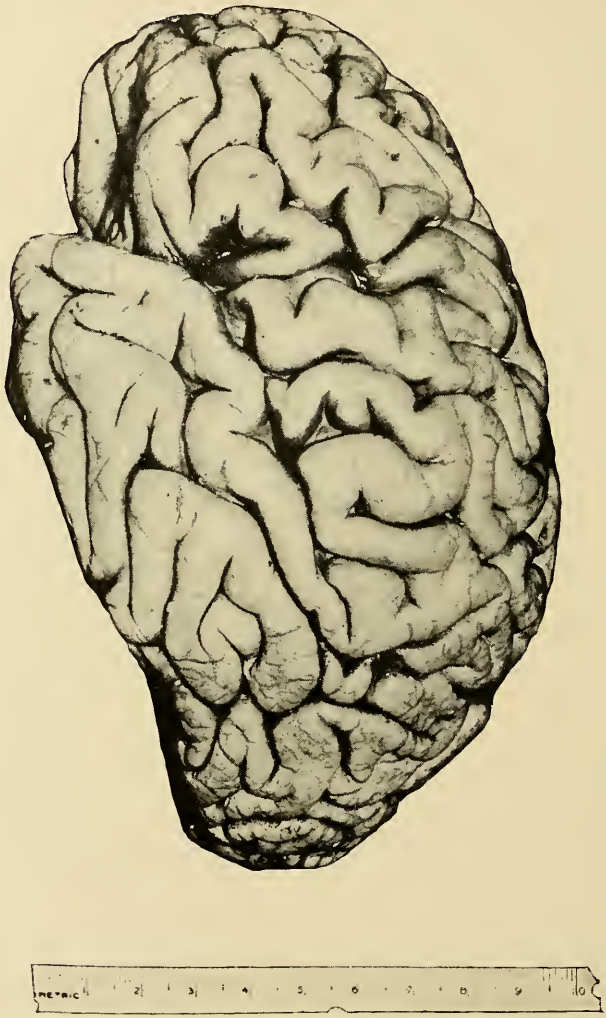

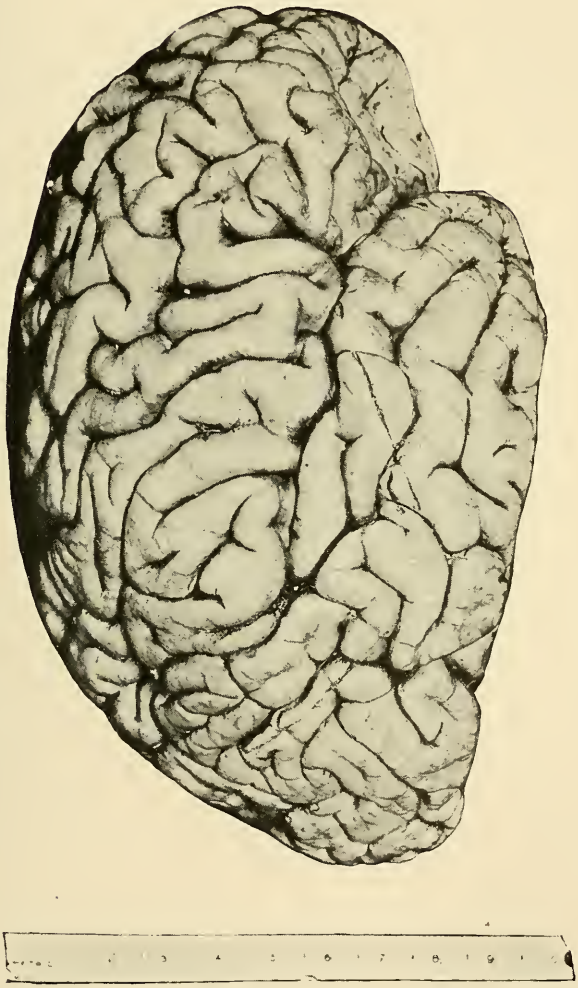

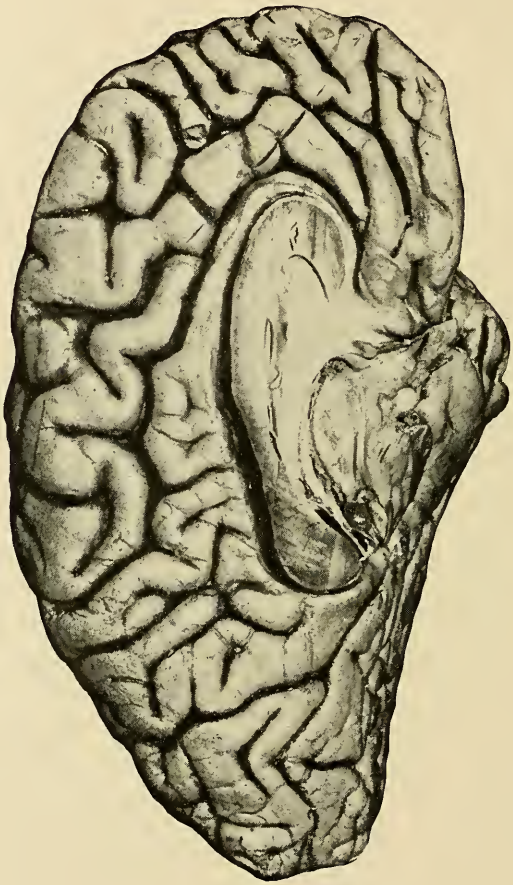

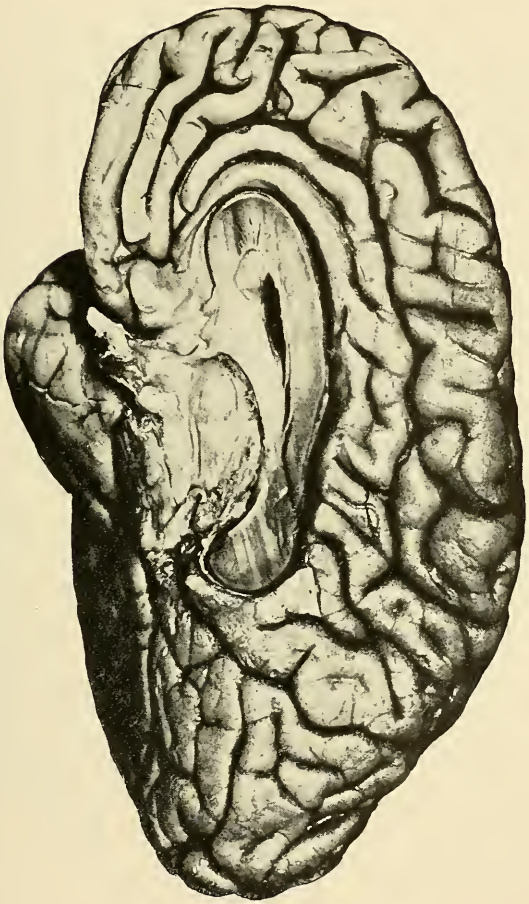

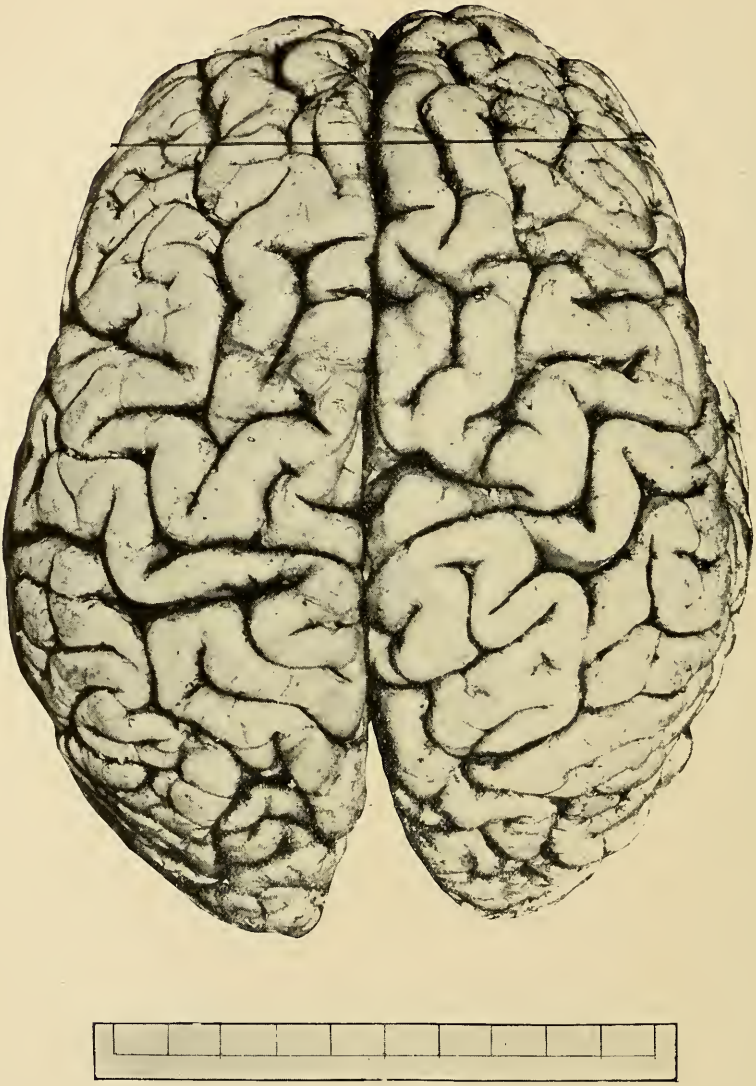

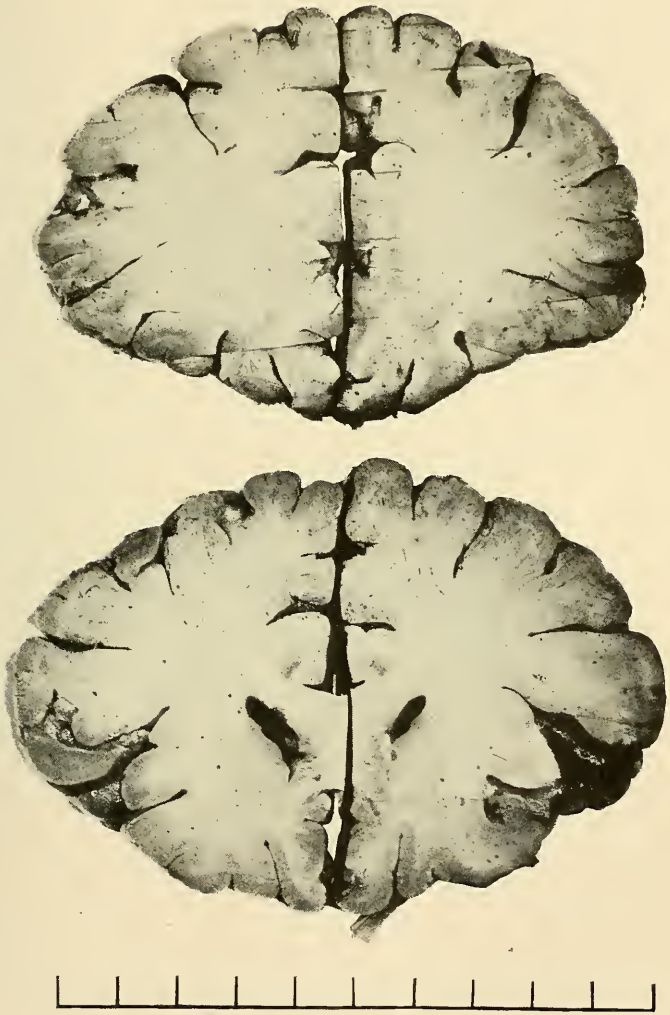

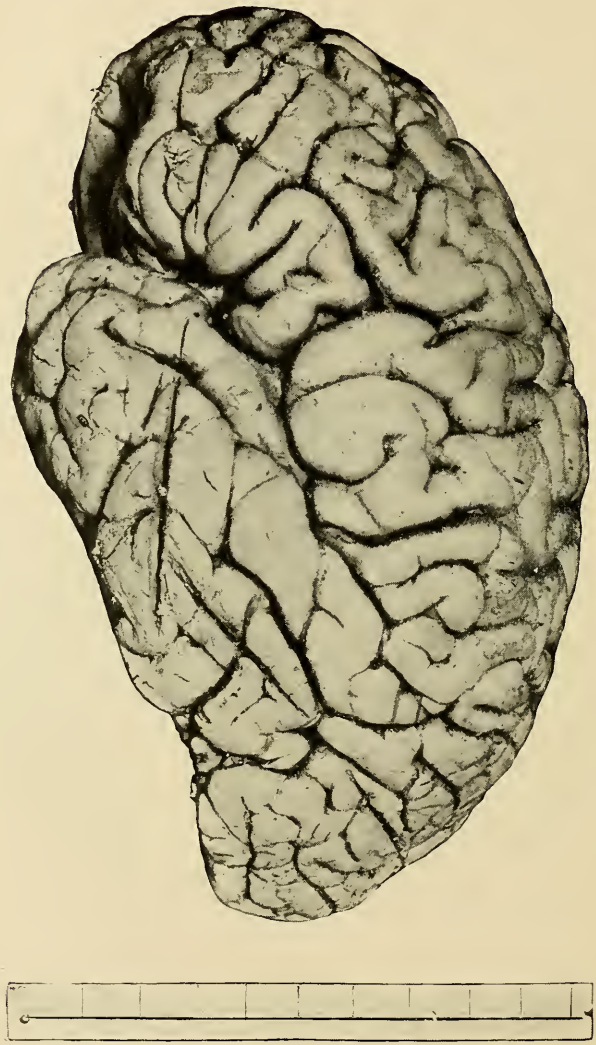

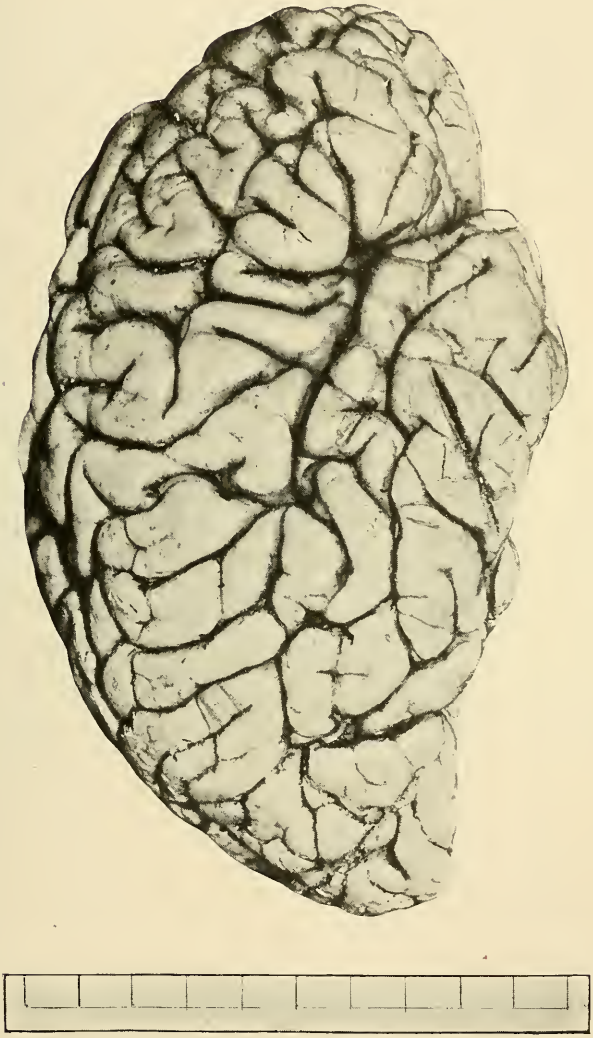

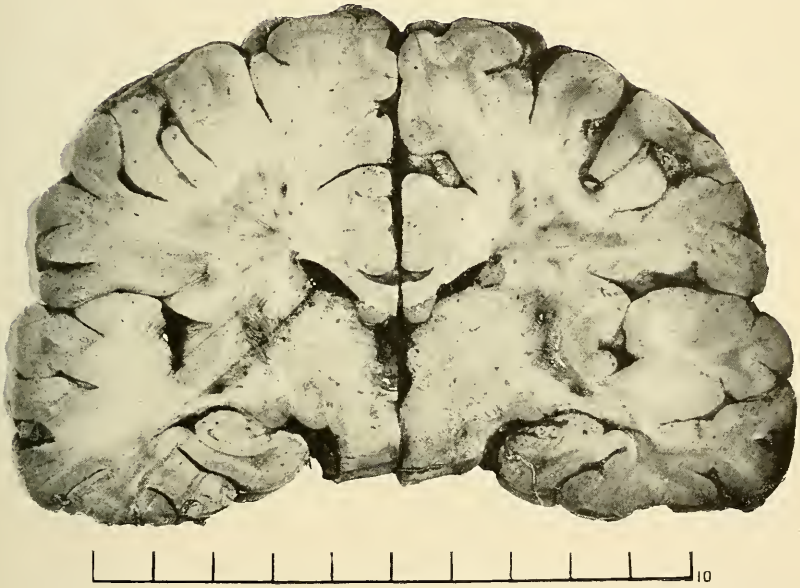
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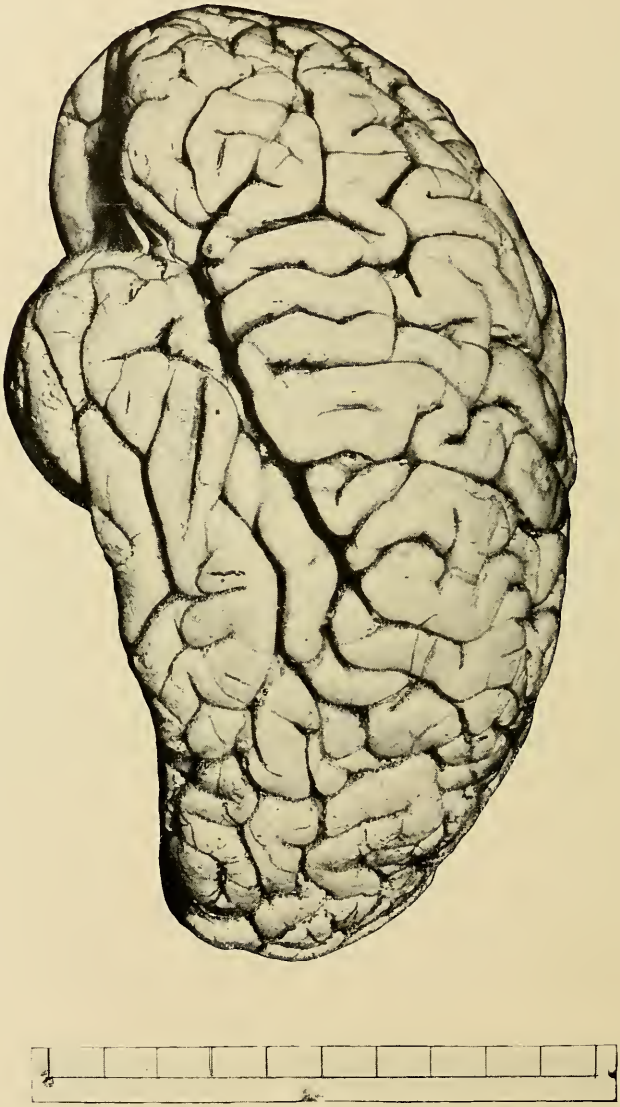

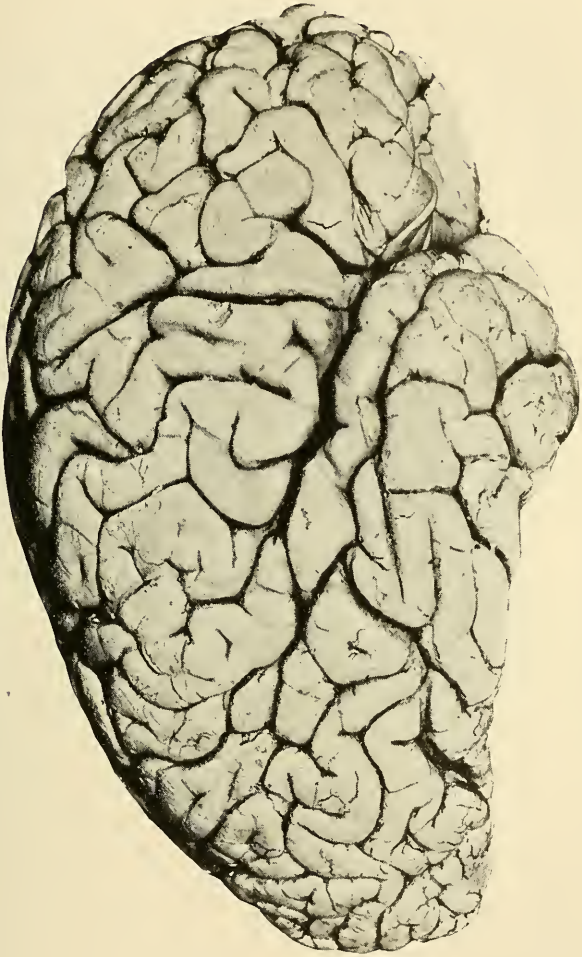

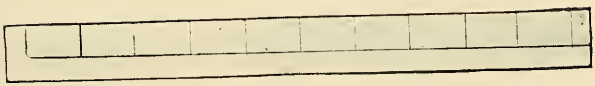

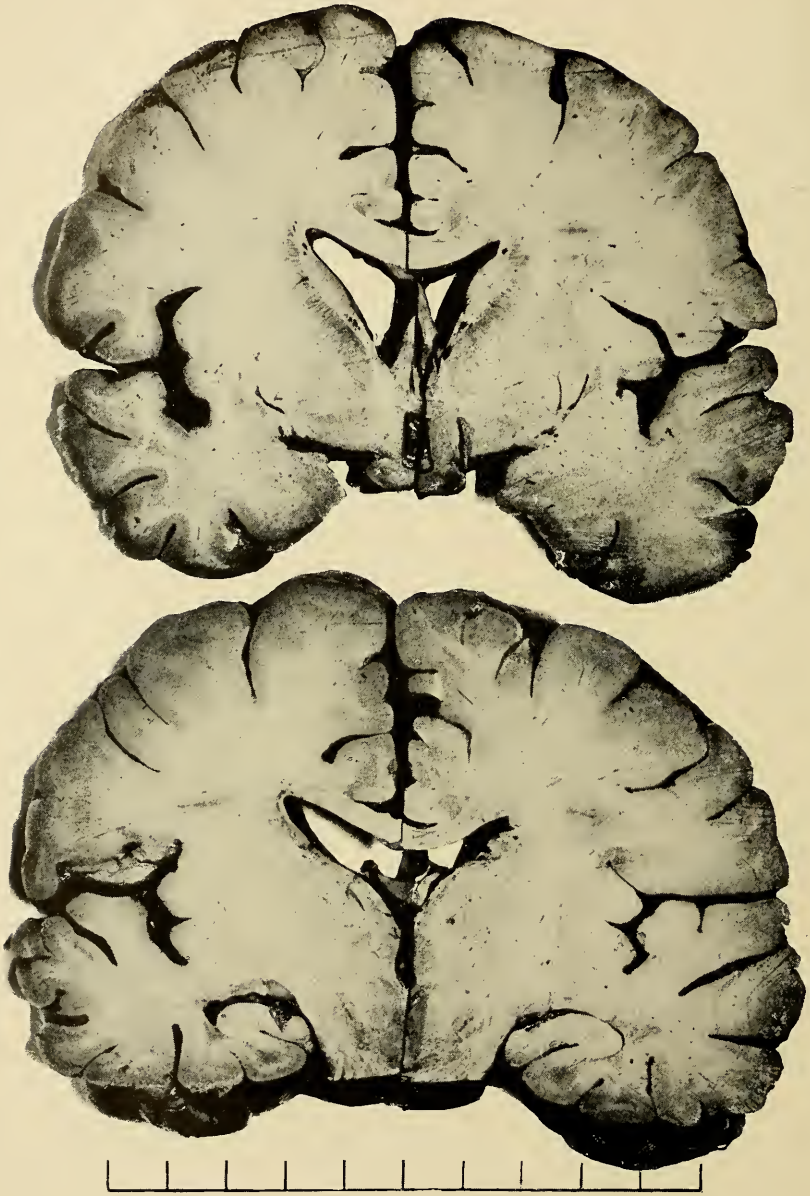

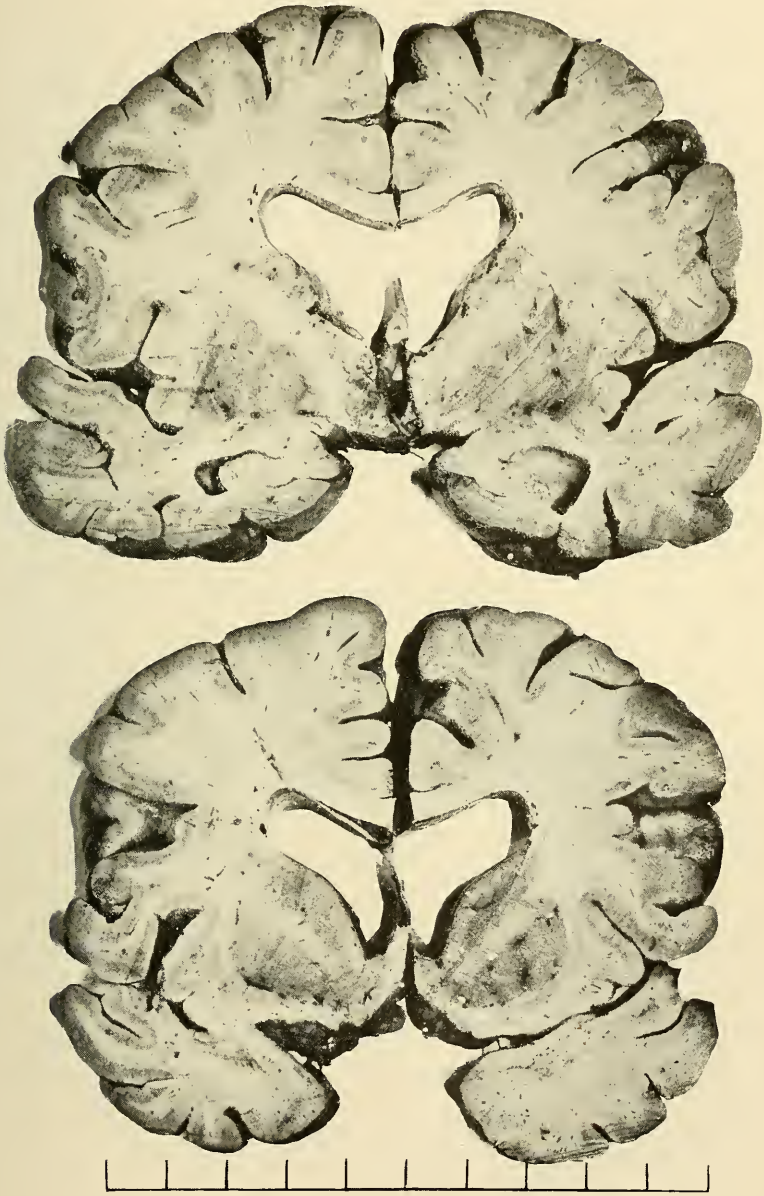

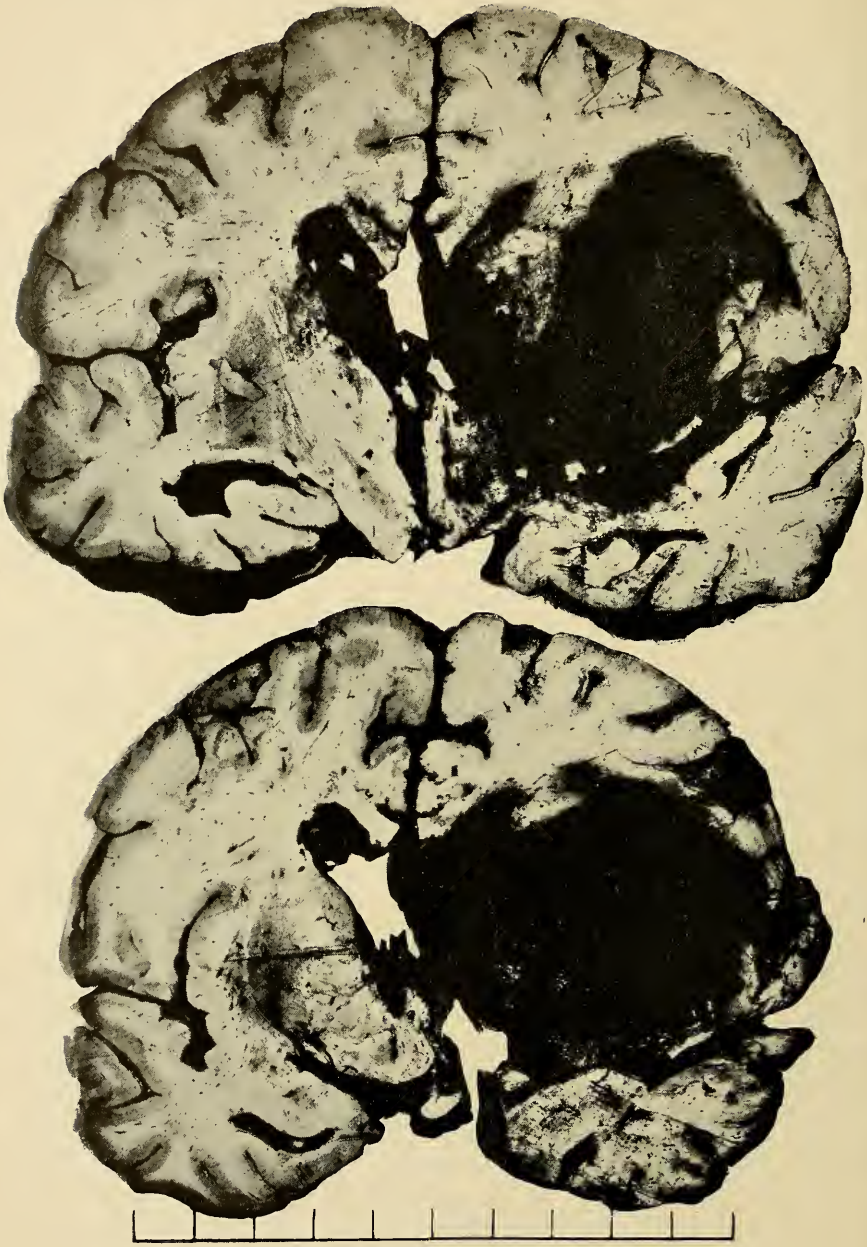

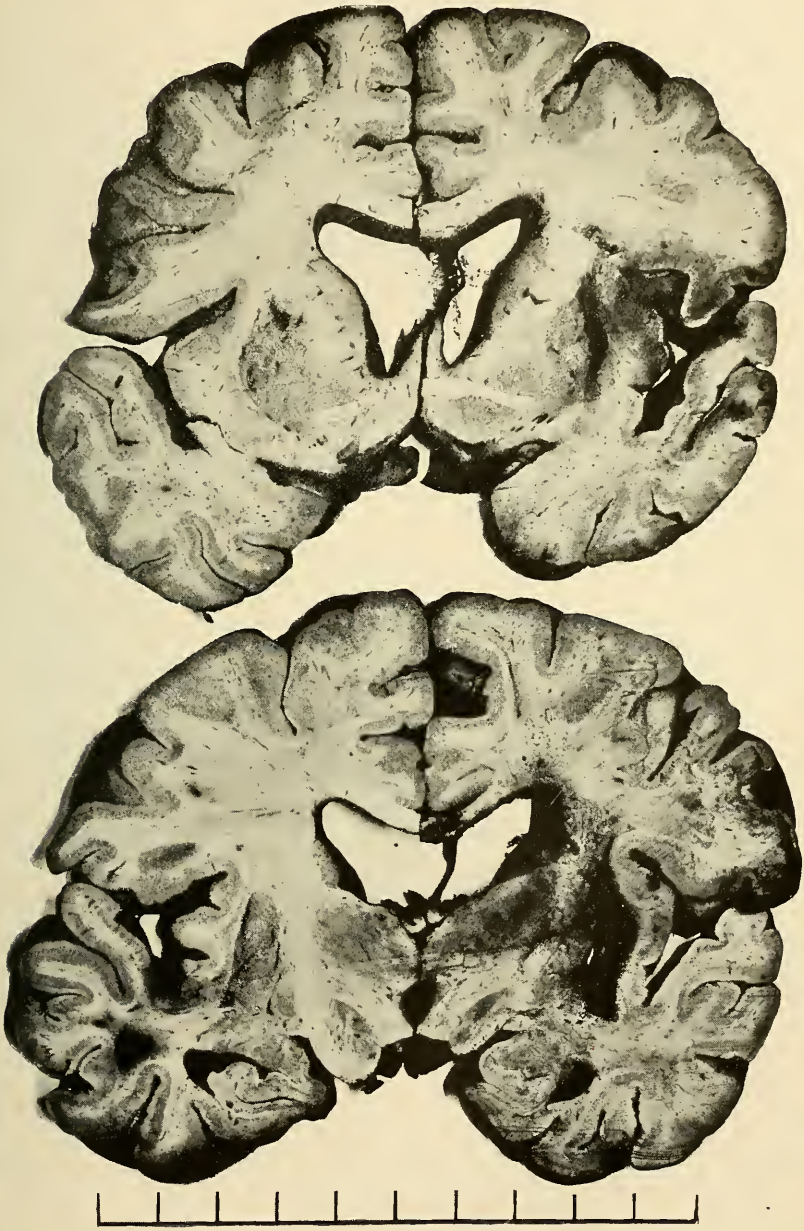

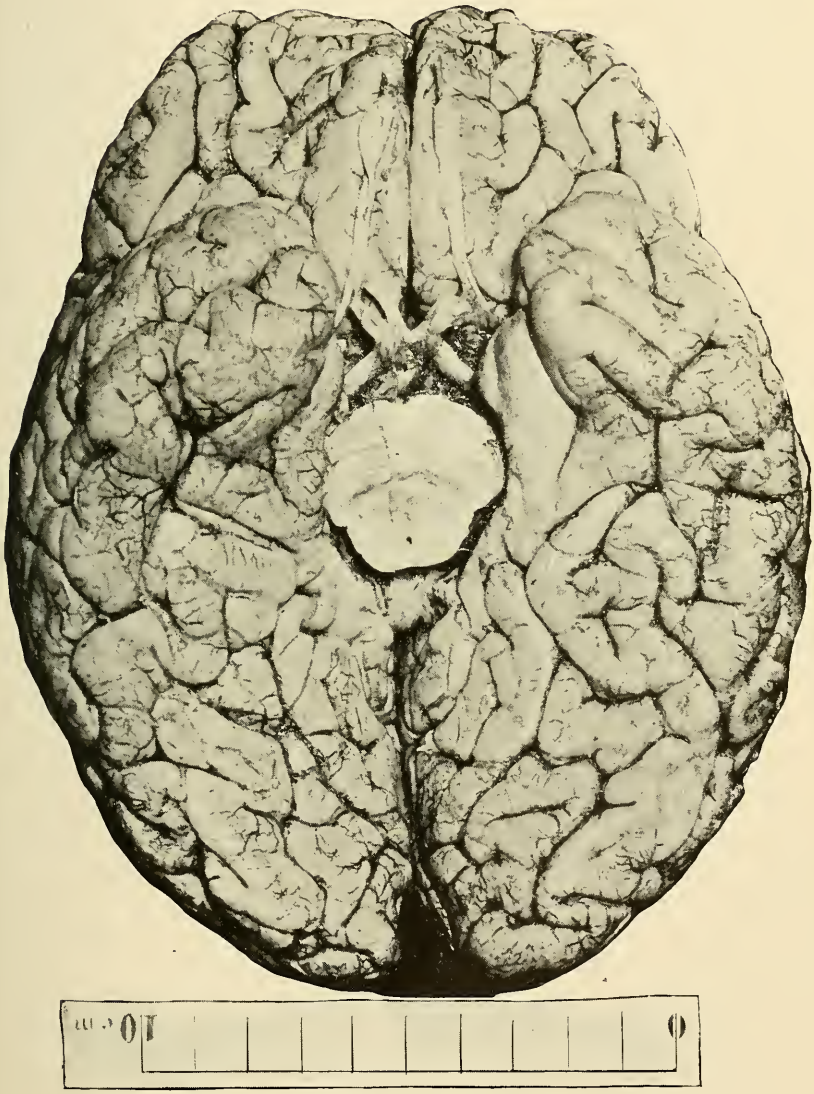

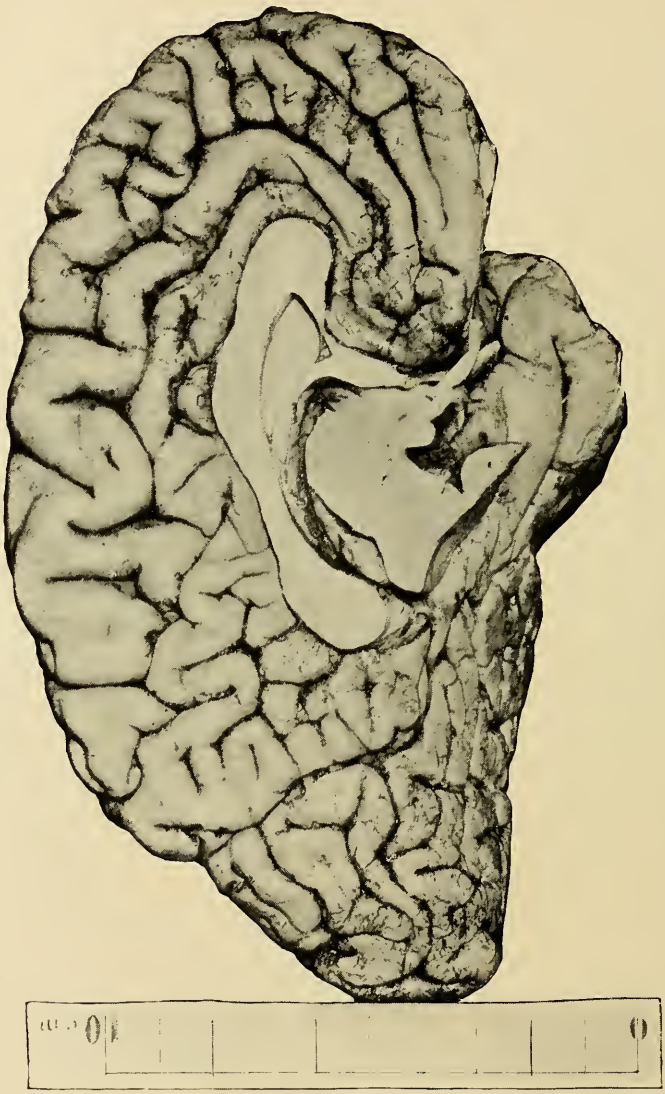

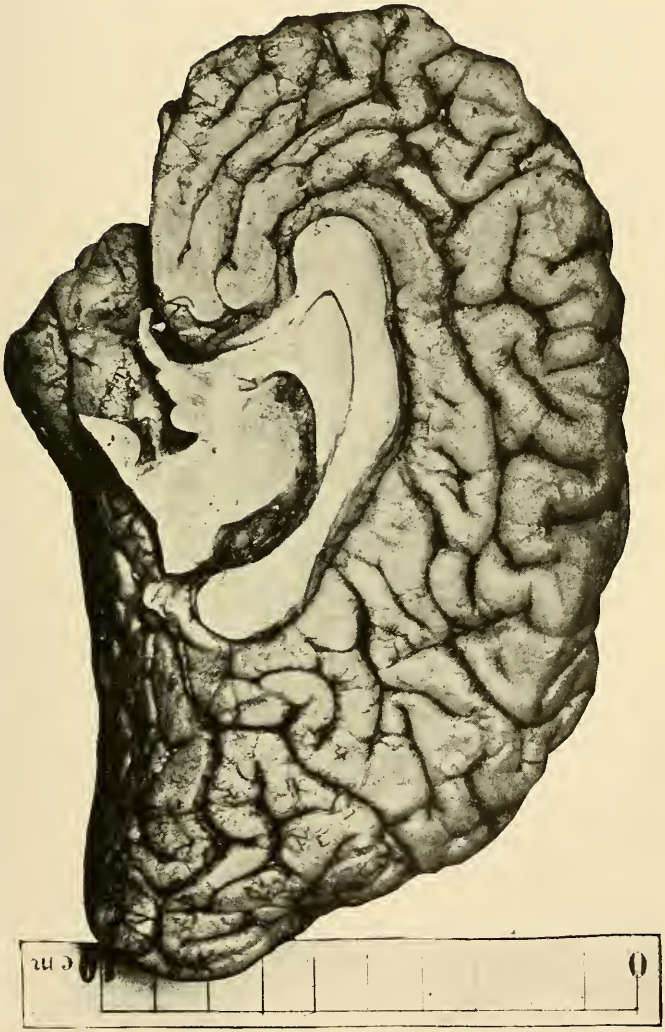

XLVI

A STUDY OF NORMAL-LOOKING BRAINS IN PSYCHOPATHIC SUBJECTS.

Third Note (Boston State Hospital Material).*

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AND

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Following is a third fragment of the study now in progress of the brain side of the so-called functional psychoses. Functional psychoses ought to be such that the brains would show nothing that should not be there, nothing not quite "physiological," nothing not essentially reversible and capable of return to the *status quo*. In order to study functional psychoses, we, strictly speaking, need to find cases without intrinsic brain changes. In the midst of the host of intrinsic and extrinsic changes which a majority of brains in psychopathic sub-

* Being Contributions of the State Board of Insanity, Number 35 (1915.1). The communication was read in abstract at the meeting of the Norfolk District Society in April, 1914. (*Bibliographical Note.*—The previous contribution was by Dr. A. W. Stearns (1914.14), entitled "Out-Patient Work in Massachusetts State Hospitals for the Insane," Boston Med. and Surg. Jour., Vol. clxxi, November 5, 1914.)

jects shows, it has proved difficult to secure an irreproachable functional series. The more exact and finical the brain examination, the harder is it to secure "normal-looking" brains, although the "changes" and "anomalies" found may have had nothing to do with the particular psychopathy of the given case.

The percentages of normal-looking brains in the series so far studied are as follows and may be considered as roughly true in consideration of the anatomical criteria prevalent in the different laboratories.

Worcester[1]	249	741
Taunton[2]	70	450
Danvers[3]	235	1000
Westborough[4]	73	500
	627	2691

To these percentages may be added that of the Monson State Hospital (for epileptics).

Monson[5]	76	205

We wish to communicate the results of an examination of 153 brains at the Boston State Hospital, in which there was, if you please, an intentional bias—a bias towards the Danvers standards of counting all suspicious findings among "abormalities." We need not repeat that we do not suppose that all the abnormalities found in the majority of this or any other series are directly correlatable with the psychopathic status of the subjects which carried the brains. What we are investigating is the normal-looking residuum.

Among these 153 were found 20 brains of a normal appearance, *i.e.* brains showing no destructive lesions, atrophic, cystic, or otherwise, and no sclerosis (in the sense of tissue indura-

tion or gliosis, regarded as of pathological nature). Some cases of basal or other arteriosclerosis and some of chronic meningeal change were counted as normal-looking, because upon careful search no sign of *substantial* brain change could be found.

Adhering in general to Kraepelinian lines in diagnosis, we find these 20 distributed as follows:—

Endogenous deteriorations (dementia precox)	2*	(xix, xx)
Manic-depressive group (3 involution cases included)	6	(xiii-xviii)
Senile and allied organic psychoses	4	(vii-x)
General paresis	1	(i)
Symptomatic psychoses	2†	(xi, xii)
Epileptic psychosis	1	(v)
Alcoholic psychoses	2	(iii, iv)
Central neuritis	1	(ii)
Syringomyelia	1	(vi)
	20	

Dementia precox forms (10 cases) almost 7% of the *total* B. S. H. series (153 cases); it forms 10% of the *normal-looking* series. One brain in five dementia precox brains is normal-looking.

Manic-depressive psychoses (involutional cases included) form (12 cases) about 8% of the *total* series; they form 30% of the *normal-looking* series.

Senile psychoses (with some allied forms) form about 29% of the total series; they form 20% of the normal-looking series.

In order to secure, if possible, a *microscopically* normal set of cases—cases not merely normal-looking in the gross, but normal-looking with the microscope—some orienting studies have been made with the microscope. These studies

* Possibly 3, owing to the possible inclusion of xii.
† Possibly 1, if xii be transferred to dementia precox.

have permitted already the solution of some problems.

I. *Normal-looking brain in general paresis (five months' duration of symptoms).* Microscopically characteristic (plasma-cells about cortical vessels, cell-losses, gliosis) in various areas. Summary follows (details, clinical and structural to be presented in another "note" in this series).

B. S. H. 10,891. Path. (1912.55). Male. Age at onset and death, 53.

Duration of mental disease, 4 months, 27 days.

Mental disease, *continuous.*

Diagnosis. Never in doubt (Psychopathic Hospital, Boston State Hospital).

Terminal Illness. Bronchopneumonia.

Trunk — Chronic Changes. Trichina spiralis, pleuritis, pericarditis.

Trunk — Acute Changes. Hemorrhage in mesentery, bronchopneumonia, bronchial lymph nodes.

Body length—172 cm. *Brain weight*—1270 grams (Tigges' formula—1376 grams).

Lymph Nodes. Not notable.

Ductless Glands: Pituitary, edematous; adrenals, one diffusely red; testes, do not thread well.

II. *Normal-looking brain in central neuritis (six weeks' duration of symptoms).* Microscopically characteristic (numerous axonal reactions in various areas) but perhaps not a pure case (since the nerve-cell changes were not all of the axonal type and indeed some seemed to show an admixture of cell-lesions). Southard and Hodskins,[6] as well as Gay and Southard,[7] had offered some evidence to show an association between the clinical entity called "central neuritis" and softness of brain. Gay and Southard went so far as to suggest that bacillus coli communis infec-

tions of blood or cerebrospinal fluid might be at the basis of the soft brain. Canavan and Southard,[8] in a paper now in press, found in another series that bacillus coli communis failed to be associated with soft brain, but they found other bacteria capable of splitting laboratory media or of abstracting water from such media.

The present case, as the protocol would show, did *not* exhibit a soft brain; but the brain was described as "tense"-feeling and as entirely filling the dura mater, being especially prominent in the central region (brain weight 1350 gms.). The frontal portion of the pia mater was slightly hemorrhagic, and all the spinal root ganglia presented a curious and unusual appearance of hemorrhage.

Culture from the cerebrospinal fluid yielded bacterium capsulatum (capsule bacillus of Pfeiffer), an organism of the aerogenes group. The blood was sterile. Culture from bronchial lymph node yielded micrococcus aureus and micrococcus roseus.

The total course of symptoms in this patient (44 years of age) was 6 weeks. There is certainly a fair question of the diagnosis. Perhaps the root-ganglion hemorrhages acted to produce reflexly the characteristic movements which are more usually found in "central neuritis."

The histological examination of the hemorrhagic root-ganglia shows hemorrhages in spaces of the dura mater adjacent to the ganglia and small hemorrhages in the peridural fatty tissue. There are no evidences of acute inflammation in the dura mater or fatty tissue. Minute vessels within the proximal portion of the dorsal roots show endothelial nuclei of vesicular and slightly swollen appearance, with at one point a moderate number of mononuclear cells of a vesicular

appearance in the adventitia. There are no good examples of lymphocytes in the tissue.

If the clinical phenomena are to be at all related to these peripheral changes, it would seem that they must be largely due to mechanical pressure changes, unless we are to interpret the very slight endothelial changes in the small vessels as toxic.

The Marchi sections show fat in cells within the hemorrhage, but do not show evidences of fat in the nerve fibers.

This case evidently deserves more particular study. For completeness' sake we present data as in other cases of this series.

B. S. H. 12,018. Path. 1913.44. Female. Age at onset and death, 44 years.

Duration before admission, 1 month and 14 days.

Hospital Residence. One day.

Diagnosis. (Melancholia (?)—by outside physicians. (Central neuritis, Boston State Hospital.)

Continued mental illness.

Terminal Illness. Acute interstitial nephritis?

Bacteriology. Heart's blood, negative.

Cerebrospinal Fluid. Bacterium capsulatum.

Bronchial Lymph Node. Micrococcus pyogenes aureus and micrococcus roseus.

Trunk—Chronic Changes. Pleuritis (right) sclerosis of coronaries and aortic valve. Fatty liver, fibroma of uterus.

Trunk—Acute Changes. Hypostatic pneumonia, peribronchial lymphnoditis, follicular colitis, interstitial nephritis.

Body Length—152 cm. *Brain Weight*—1350. (Tigges' formula 1216.)

Lymph Nodes. Peribronchials enlarged.

Ductless Glands: Pituitary negative; thyroid small; adrenals thin. Ovaries firm.

III. *Normal-looking brain in Korsakoff's (alcoholic) psychosis (ten weeks' duration of*

symptoms). *Erroneously diagnosticated general paresis.* The erroneous diagnosis was at the Psychopathic Hospital, whose officers chose to discount a negative Wassermann serum test and a negative cytological examination of the liquor cerebrospinalis, and trusted to certain suggestive mental symptoms and reflex-disorders. Upon transfer to the Main Hospital, the case was diagnosticated Korsakoff's psychosis, on the basis of the following findings: Pupils unequal, reacting to light within narrow limits, and are irregular in outline. Knee jerks, right more lively than left; trouble in locomotion; restlessness; hunger; untidiness; fabrication and miscalling of bystanders.

B. S. H. 11,226. Path. 1913.6. Male. Age at death, 44.

Duration. Two months, 14 days.

No previous attacks.

Terminal Illness. Sudden death, choking and vomiting.

Bacteriology.—Heart's blood negative.

Cerebrospinal Fluid. Cladothrix invulnerabilis.

Trunk—Chronic Changes. Fibrous pleuritis, epicarditis, sclerosis coronaries, endocarditis, aortic sclerosis, pulmonary tuberculosis. Horse-shoe kidney.

Trunk—Acute Changes. Ileitis, injection duodenum.

Body Length—168 cm. *Brain Weight*—1450. (Tigges' formula 1344.)

Lymph Nodes. Negative.

IV. *Normal-looking brain (over-small?) in delirium tremens (five weeks' duration of symptoms) erroneously diagnosticated general paresis, tabetic form.* Microscopic examination showed numerous acute cell changes, but no evidence of general paresis.

It is probable that this brain, weighing but 1010 grams as against a weight calculated by Tigges' formula (8 x 151) (the body length) of 1208 grams, should be regarded as either hypoplastic or atrophic.

B. S. H. 9,981. Path. (1911.11). Female. Age at death, 35.

Duration of Mental Illness. One month, 4 days. Continuous mental disease.

Diagnosis. Taboparesis (erroneous).

Bacteriology. Heart's blood, streptococci. Cerebrospinal fluid, negative. Right middle ear, bacterium aerogenes group. Mesenteric lymph node, negative.

Trunk—Chronic Changes. Decubitus. Splanchnoptosis, ascites, cystic oöphoritis, epicarditis, hypertrophy of heart. Healed pulmonary tuberculosis, chronic interstitial nephritis, chronic ileitis, uterine growth.

Trunk—Acute Changes. Hemorrhagic-oöphoritis, acute diffuse nephritis, injection lower lobes of lungs.

Lymph Nodes. Mesenteric enlarged.

Ductless Glands. Pituitary negative. Thyroid very small. Ovaries—one cystic, one hemorrhagic, adrenals normal.

Body Length—151 cm. *Brain Weight*—1010 grs. (Tigges' formula 1208.)

V. *Normal-looking brain in epileptic psychosis.* (Epilepsy since infancy, dementia from 18+, death at 38.) Microscopically, numerous cell losses,—not here further considered.

B. S. H. 4810. Path. (1911.26). Male.

Diagnosis. Epilepsy.

Terminal Illness. Bronchopneumonia.

Trunk—Chronic Changes. Emaciation, hypertrophy of heart, decubitus.

Trunk—Acute Changes. Bronchopneumonia, congestion kidneys.

Lymph Nodes. In left groin enlarged.

Ductless Glands. Pituitary, thyroid, adrenals negative, testes negative.

Body Length—150 cm. *Brain Weight*—1460 grs. (Tigges' formula 1200)

VI. *Normal-looking brain in a syringomyelic subject with mental symptoms.* (Special study to be published shortly by Dr. H. I. Gosline.)

B. S. H. 9445. Path. (1911.31). Male. Age at onset of mental symptoms, 39 years. Age at death, 43 years.

Duration of Mental Disease. 42 months, 27 days. Mental disease uninterrupted.

Diagnosis. Organic dementia or general paresis.

Terminal Illness. Acute vegetative endocarditis.

Trunk—Chronic Changes. Decubitus, pericarditis, endocarditis, aortitis, fatty hepatitis.

Trunk—Acute Changes. Acute vegetative endocarditis.

Body Length—162 cm. *Brain Weight*—1355 grs. (Tigges' formula 1296 grs.)

Lymph Nodes. Not enlarged (bronchial).

Ductless Glands. Pituitary, thyroid, adrenals, negative. Testes negative.

VII-X. *Four normal-looking brains in Senile Psychoses.* The microscopic examination of all four, so far as completed, exhibits obvious cell-losses, although three of the brains are considerably overweight, and two of them markedly so.

B. S. H. 9314. Path. (1911.32). Female. Age at onset, 64. Age at death, 66.

Duration. 25 months, 10 days.

Continuous mental disease.

Diagnosis. Senile dementia, senile psychosis (presbyophrenic type).

Terminal Disease. Chronic interstitial nephritis, cardiac hypertrophy and general anasarca.

Bacteriology. Heart—micrococcus xanthogenicus, micrococcus varians, micrococcus rubescens. C.S.F. —micrococcus xanthogenicus, micrococcus luteus, micrococcus rubescens, micrococcus simplex.

Retroperitoneal Lymph Node. Micrococcus pyogenes aureus.

Trunk—Chronic Changes. Slight emaciation, ascites, periappendicitis, hydropericardium, myocarditis, endocarditis, apical tuberculosis (healed), pleuritis, nephritis, perihepatitis, gall stones, passive congestion liver.

Trunk—Acute Changes. Absent.

Lymph Nodes. No change.

Ductless Glands. Pituitary, thyroid, negative; adrenals negative, ovaries cystic.

Body Length—164 cm. *Brain Weight*—1340 grs. (Tigges' formula 1312).

B. S. H. 10.500. Path. (1912.37). Female. Age 71.

Duration of Mental Disease. Seven months, 19 days. Mental disease continuous.

Diagnosis. Senile psychosis.

Terminal Illness. Bronchopneumonia.

Trunk—Chronic Changes. Emaciation, adhesive pleuritis, bronchiectasis, brown atrophy of heart, chronic interstitial nephritis, myoma of uterus, hepatic atrophy, splenic atrophy, arteriosclerosis.

Trunk—Acute Changes. Streptococcus septicemia.

Ductless Glands. Adrenals large.

Body Length—153 cm. *Brain Weight*—1380 grs. (Tigges' formula 1224 grams.)

B. S. H. 10,192. Path. (1913.16) Male. Age at onset, 75. Age at death, 77.

Duration. 21 months, 21 days.

Diagnosis. (Senile psychosis with delusions and deterioration.) (Cerebral arteriosclerosis.)

Terminal Disease. Gangrene of lung and arteriosclerosis.

Bacteriology. Heart's blood micrococcus tenacatus. Cerebrospinal fluid, bacillus formosus.

Trunk—Chronic Changes. Elephantiasis? decubitus, ascites; sclerosis mammary, coronary arteries; thrombosis mesenteric veins; pleuritis, aortitis, healed pulmonary tuberculosis, renal lithiasis, hepatic cysts, hypertrophy of prostate, lipoma.

Trunk—Acute Changes. Fibrinous pleuritis, gangrene of lung, hemorrhages in spleen.

Lymph Nodes. Perioesophageal lymph nodes enlarged. Peribronchial lymph nodes enlarged.

Ductless Glands. Pituitary, adrenals negative.

Body Length—137 cm. *Brain Weight*—1440 grs. (Tigges' formula 1096 grms.)

B. S. H. 10,241. Path (1911.42). Female. Age at onset, 77. Age at death, 80.

Duration—36 months, 22 days.

Continuous mental disease.

Diagnosis. Senile psychosis, simple deterioration.

Trunk—Chronic Changes. Emaciation, edema, decubitus, periappendicitis, perihepatitis, sclerosis majority of vessels. Hypertrophy heart, endocarditis, pulmonary tuberculosis, interstitial nephritis, fibromata of uterus.

Trunk—Acute Changes. Hemorrhagic ascites.

Ductless Glands. Pituitary negative, thyroid small, adrenals plump and large.

Body Length—140 cm. *Brain Weight*—1125 grs. (Tigges' formula 1120 grams.)

XI. *Normal-looking brain in an exhaustion-psychosis of unknown but brief duration.* Orienting microscopic examination of the two prefrontals, superior parietal and calcarine areas, showed cell losses characteristically in the outer layers, but also to some extent in inner layers. The most marked cell losses appear in prefrontals and left superior parietal area. It seems hard to explain these findings on the basis of a lesion of brief duration unless at times a brief or critically acting agent may kill cells in such wise as to provoke little or no neuroglia reaction.

B. S. H. 10,284. Path. (1912.7). Male, aged 43. Age at onset, 42. In Hospital 2 months, 25 days.

Duration before admission, unknown.

Diagnosis. Exhaustion psychosis.

Terminal Illness. Pulmonary tuberculosis.

Bacteriology. Heart's blood, micrococcus cumulatus. Cerebrospinal fluid, micrococcus ovalis.

Trunk—Chronic Changes. Sacral decubitus, chronic periappendicitis, sclerosis coronaries, hypertrophy heart, interstitial nephritis, atrophy of liver.

Trunk—Acute Changes. Bronchopneumonia.

Ductless Glands. Adrenals fat. Pituitary edematous. Undescended testicle.

Lymph Nodes. Mesenteric lymph nodes prominent.

Body Length—141 cm. *Brain Weight*—1550 grs. (Tigges' formula 1128 grams.)

XII. *A doubtful case of paranoiac nature, complicated by toxic features.* The orienting sections showed only slight and focal cell losses in the cortex, and for the rest only various marked evidences of cell disease without cell destruction.

B. S. H. 10.416. Path. (1912.41). Female. Age at onset, 50? marked at 59. Age at death, 60.

Duration of Mental Disease. 127 months, 27 days. Mental disease continuous.

Diagnosis. Paranoiac condition. (Toxic?)

Terminal Illness. Pulmonary tuberculosis.

Trunk—Chronic Changes. Emaciation, visceral peritonitis, pleuritis, pulmonary tuberculosis, pericarditis, hypertrophy of heart. Tuberculosis of intestine (ulceration) perioöphoritis.

Trunk—Acute Changes. Thrombus, aorta.

Lymph Nodes. Bronchial and mesenteric enlarged.

Ductless Glands. Pituitary, thyroid, negative; adrenals small, ovaries small.

Brain Weight—1130 grams. (Tigges' formula 1184 grams.)

XIII-XV. *Three normal-looking brains in cases of involution-psychoses.* Orienting examination in these cases similar to XI indicated that XIII was probably a case of brain atrophy, that XIV was possibly an arteriosclerotic dement, (at any rate microscopic changes in this patient of 84 were rich enough) and that XV was quite beyond question a case of brain atrophy.

B. S. H. 7771, 8933. Path. (1911.5). Female. Age 71. Age of onset, 65; age at death, 73.

First Attack. Duration (onset 65), 24 months, 12 days.

Second Attack. Duration (onset 70), 31 months, 11 days.

Diagnosis. Chronic melancholia.

Terminal Disease. Facial erysipelas and bronchopneumonia.

Bacteriology. Heart's blood, bacterium gallinarium. Cerebrospinal fluid: Micrococcus candicans.

Trunk—Chronic Changes. Emaciation, ascites, splanchnoptosis, periappendicitis, pulmonary tuberculosis, pleuritis, coronary sclerosis, endocarditis, gall stones, interstitial pancreatitis, fibromata oöphoritis.

Trunk—Acute Changes. Hemorrhagic endometritis, bronchopneumonia.

Lymph Nodes. Cervical lymph nodes calcified.

Ductless Glands. Pituitary negative, thyroid—cystic, adrenals, autolysed, ovaries atrophied.

Brain Weight—1130 grams. (Tigges' formula 1240.)

B. S. H. 4798, 5394. Path. (1912.11). Male. (1) Age at onset, first, 67. In hospital Aug. 2, 1895, to Dec. 16, 1895. Discharged improved.

Diagnosis. Acute melancholia.

(2)Age at onset, second, 70. In hospital, 14 yrs.

Diagnosis. Involution melancholia.

Entire duration of mental disease, 168-3/30 mo.

Cause of Death. Acute dilatation of the heart.

Course of Disease. Interrupted at first, latterly continuous.

Bacteriology. Heart's blood. Micrococcus cremoides. Cerebrospinal fluid, negative. Bladder, bacterium gallinarum. Prostate, bacterium gallinarum.

Trunk—Chronic Changes. Perisplenitis, peri- and hepatitis. Sclerosis—mammary, coronaries and aorta. Fatty myocarditis, hepatitis. Dilatation of heart and two incompetent valves, tricuspid and mitral. Chronic interstitial nephritis with cysts.

Acute Changes. Injection gastric and intestinal mucosa, acute cystitis, pus in prostate—acute splenic tumor.

Lymph Nodes. None enlarged.

Ductless Glands. Thyroid, adrenals very small. Testes, pituitary not notable.

Body Length—172 cm. *Brain Weight*—1430 grs. (Tigges' formula, 1346 grams.)

B. S. H. 9924. Path. (1912.29). Female. Age at onset, 58; in hospital 18-14/30 mo.

Entire length of mental disease, 20-14/30 mo.

Diagnosis. Involution melancholia.

Course of Disease. Continuous.

Terminal Illness. Septic endocarditis with multiple septic emboli.

Trunk—Chronic Changes. Chronic pleuritis, hypertrophy of heart, sclerosis aorta, atrophy of spleen, atrophic uterus, splenic infarction.

Trunk—Acute Changes. Slight hemorrhages in gastro-intestinal tract, thrombus in aorta.

Lymph Nodes. Not enlarged.

Ductless Glands. Adrenals, ovaries, pituitary negative. Testes negative.

Body Length—150 cm. *Brain Weight*—1050 grs. (Tigges' formula, 1200 grams.)

XVI-XVIII. *Three normal-looking brains in manic-depressive psychosis.* Orienting examination in these three cases showed in XVI and XVIII a moderate degree of cell loss and in XVII nu-

merous cell losses, especially in the upper layers. Possibly XVI and XVIII are most nearly normal microscopically of this whole group (with the exception of the dementia precox XX).

B. S. H. 9483. Path. (1911.36). Female. Age at onset, 29 years. Age at death, 31 years.

Duration. One month before admission. Total duration, 19 months, 28 days.

Diagnosis. Manic depressive.

Terminal Disease. Pulmonary tuberculosis and enteritis.

Trunk—Chronic Changes. Emaciation, edema, decubitus, ascites, pleuritis, pericarditis, hydropericardium, heart hypertrophy, sclerosis aorta, anomaly aorta. Pulmonary tuberculosis, intestinal tuberculosis, interstitial nephritis, fatty hepatitis, cystic organ of Rosenmüller.

Trunk—Acute Changes. Absent.

Lymph Nodes. Induration mesenteric lymph nodes.

Ductless Glands. Pituitary plump, adrenals soft, ovaries firm and thin.

Body Length—152 cm. *Brain Weight*—1140 grs. (Tigges' formula, 1216 grams.)

B. S. H. 5,884, 6,791, 7,040, 10,594. Path. (1913.29). Female.

(1) Age at onset, 41; in hospital 2 years. Discharged improved. *Diagnosis.* Acute melancholia.

(2) Age at onset, 43; in hospital 4 months. Discharged improved. *Diagnosis.* Folie circulaire.

(3) Age at onset, 43; in hospital 7 years. Discharged to Medfield. *Diagnosis.* Recurrent mania. Manic depressive insanity. In Medfield two years, discharged to Boston State Hospital.

(4) Fifty-two years. In hospital 13 months. Entire duration mental disease, 162-17/30 mo.

Diagnosis. Manic depressive.

Interrupted at first, latterly continuous.

Terminal Illness. Pulmonary tuberculosis.

Bacteriology. Heart's blood, negative. Cerebrospinal fluid negative.

Trunk—Chronic Changes. Emaciation, atrophy of heart, fatty liver, pulmonary tuberculosis, tuberculosis of intestines, lymphatic hyperplasia.

Trunk—Acute Changes. Absent.

Lymph Nodes. Lymph node hyperplasia appendix and cecal nodes. Ductless glands, adrenals negative, ovaries elongated, white, flat.

Body Length—153 cm. *Brain Weight*—1300 grs. (Tigges' formula 1224).

B. S. H. 11.312. Path. (1913.7). Female.

First Attack. Age at onset 20 (for one year). Recovered, not in a hospital.

Second Attack. Age at onset, 62 (for two years, 14 days). Age at death, 64.

Diagnosis. "Unclassified," Psychopathic Hospital. Manic depressive, depressed; second attack, Main Hospital.

Terminal Illness. Bronchopneumonia. Several days' duration.

Bacteriology. Heart's blood, streptococcus acidi, lactici. Cerebrospinal fluid, bacillus cinctus.

Trunk—Chronic Changes. Corset liver, pleuritis, sclerosis, aorta, coronaries; atrophy of spleen, interstitial nephritis, gall stones, hydropericardium, thickening and swelling right vocal cord, bronchopneumonia, lymphnoditis, vegetative aortitis, pial hemorrhages, aortic thrombus.

Lymph Nodes. Peribronchial enlarged.

Ductless Glands. Adrenals negative, ovaries sclerosed.

Body Length—148 cm. *Brain Weight*—1370 grs. (Tigges' formula 1184.)

XIX-XX. *Two normal-looking brains in dementia precox.* Case XIX, with a well marked underweight brain showed also an underweight heart and liver but failed to show any marked evidence of cell loss in orienting examination as carried out in the six selected regions as mentioned under XI. Only after considerable search in further section was any evidence of cell loss discovered, namely foci of cell loss in right second temporal gyrus. Naturally it would be

unwise to correlate the clinical symptoms of any case with so exiguous a lesion.

Case XX showed moderate degrees of cell loss in practically all the areas examined. As above stated, Case XX and the manic-depressive cases, XVI and XVIII are the most free of microscopic alterations of a marked degree in the series.

B. S. H. 8663. Path. (1910.9). Female. Age at onset, 42. Age at death, 56.

Duration of Mental Disease. Fourteen years.

Mental disease interrupted.

Diagnosis. Dementia precox.

Terminal Illness. Osteomyelitis.

Trunk—Chronic Changes. Emaciation, decubitus, edema, ascites, pleuritis, hydropericardium, brown atrophy of heart. Endocarditis, thyroiditis, perisplenitis, gall stones.

Trunk—Acute Changes. Splenitis, nephritis, ileitis.

Lymph Nodes. Retroperitoneal lymph nodes. Superficial lymph nodes enlarged.

Ductless Glands. Ovaries atrophied. Pituitary negative. Thyroid atrophied. Adrenals negative.

Brain weight—1100 grams. (Tigges' formula 1200.)

B. S. H. 10,144. Path. (1912.47). Female. Age at onset, 25; in hospital 27 months, 27 days.

Diagnosis. Dementia precox (catatonia). Entire duration mental disease, 28-3/30 mo.

Continuous mental disease.

Terminal Illness. Pulmonary tuberculosis.

Bacteriology. Heart's blood, bacillus antenniformis. Cerebrospinal fluid, micrococcus citreus and micrococcus concentricus.

Trunk—Chronic Changes. Emaciation, edema, decubitus, tuberculous peritonitis, pulmonary tuberculosis, splenic tuberculosis, hepatic tubercles, oöphoritis and salpingitis, fibroma of uterus, hydrothorax, hydropericardium.

Trunk—Acute. Changes absent.

Lymph Nodes. Mesenteric enlarged.

Ductless Glands. Thyroid, adrenals, ovaries, pituitary negative.

Body Length—148 cm. *Brain Weight*—1210 grs. (Tigges' formula, 1184 grams).

CONCLUSIONS.

1. The present is a fragment from more extensive studies tending to settle the question how far mental disease is consistent with normality of brain; and as in previous work from the Worcester State Hospital, so this work from the Boston State Hospital has chosen to begin with normal-looking brains, since these are more likely to be essentially normal than those brains which yield obvious lesions.

2. On comparison with the Worcester percentage of normal-looking brains, viz., about one in three, and the Danvers percentage, viz., about one in four, the present Boston percentage is much lower, viz., about one in eight.

3. We do not deny that some of the lesions found in the *abnormal* brains may have had little or nothing to do with the mental disease which their bearers showed; the point of our research lodges in the endeavor to discover essentially normal brains in subjects of mental disease. There are 20 in 153 examined by uniform methods which gave promise of being microscopically as well as macroscopically normal.

4. One normal-looking brain yielded a chronic-looking exudate, Case 1 (12.55) which was a case of general paresis of brief duration (less than five months), clinically certain, showed nerve-cell, and fibre-changes, gliosis and perivascular mononucleosis (including plasma cells) of fairly even degree throughout sections examined. The gross examination yielded opaque points of thickening in the pia mater over the vertex. The dura had begun to thicken and the calvarial diploe had begun to disappear.

The brain had not lost more than 100 grams in weight (Tigges' formula).

5. One case yielded evidences of acute perivascular exudate post-pneumonic encephalitis) but the mental picture cannot be regarded as due to the exudate.

6. The suspicion is often uttered that cases not infrequently show fine vascular disease not evident in the gross. No such case has appeared; but there was one (x, 11.42) which, despite coarse changes in the basal vessels, was included in the normal-looking series and microscopically showed slightly marked fine vascular changes with equally marked cortical changes (no infarcts but generalized and focal losses). This case was a female of 80 whose brain weighed 1125 grams, *i. e.* 5 grams above the calculated weight according to body length. It is possible that the brain was slightly edematous—vacuoles among nerve cells (18 hours postmortem, tuberculous peritonitis). The brain was included in the normal-looking series, although on the autopsy table the diagnosis of "general cerebral gliosis" was made (confirmed by the excess of cells in the plexiform layer in virtually every region examined). It may be inquired why a case with basal vascular disease should not be forthwith excluded on the ground that fine changes will be certain to be found; but they are not sure to be found as XIV, 12.11, proved (since in this case there were *gross arterial* changes and *few or no fine* vascular changes).

7. We have accordingly reduced our 20 normal-looking cases to 18 which still give some promise of proving normal on microscopic examination. One of these 18 was a case of epilepsy, v, 11.26, with dementia; and, since the epilepsy began in infancy, it is doubtful whether it should be included in this study. Micro-

scopically in any event, there were numerous evidences of cell-losses.

8. If we exclude this case of epilepsy from the normal-looking numerator of the fraction, we should also possibly exclude 5 other epileptics (or in all 6) from the denominator, yielding a percentage of 11.5 *i.e.* 17 in 147 cases excluding all epileptics and two cases I and X in which the microscope revealed changes which should theoretically yield gross lesions.

9. In a study of the percentage of normal-looking brains, it would be wise also to exclude clear cases of imbecility, of which there were 2 in the series, neither of which yielded a normal-looking brain; this makes a percentage of 11.7% normal-looking brains in a series of 145.

10. In the analysis of this residue of 17 normal-looking brain cases, we must first consider the question of atrophy or aplasia. Eleven cases yielded brain weights above normal, employing Tigges' formula (*i.e.* 8 × body length in cm. = probable brain weight). Six remaining brains weighed less than normal according to this formula. Of these six, one (Case IV, 11.11) yielded a brain weight, 1010 grams, calculated weight 1208 grams, which should probably make this case fall into the atrophic brain group. The reflex picture and certain other clinical features gave rise to the diagnosis *taboparesis*. The total duration was but one month and four days. The absent knee jerks proved due either to axonal anterior horn cell reactions or to peripheral neuritis (abundant Marchi degenerations); and it is probable that we are dealing with a *Korsakoff's psychosis* (history of previous attacks of alcoholic mental disease not obtained, but possible). Abundant evidence of cell-loss with satellitosis was found in many areas microscopically.

Another case (XV, 12.29) yielded a brain weighing 1050 grams, *i.e.* a calculated loss of 150

grams. This case showed various evidences of atrophy in other organs also, and microscopically a remarkably diffuse cell loss in the cortex. Clinically the case was one of involution melancholia, 59 years of age, of 20 months and 14 days.

12. Case XIII, 11.5, with a calculated brain weight loss of 120 grams was a female of 73 years, with total duration of about 8 years. The microscopic evidences of cell loss were such that this case also must probably be placed in the atrophic group; in point of fact her brain atrophy was probably obscured by increase of weight 8½ days post mortem (brain not palpably soft on account of gliosis).

13. XIX, 10.9, with a calculated brain weight loss of 100 grams, showed a small heart (145 grams) and a small liver (1000 grams). This case probably does not belong in the atrophic group, since microscopically there was small evidence of cell-loss. This case of paranoid dementia precox will be considered below. XVI, 11.36, and XII, 12.41, with calculated brain weight losses of 76 and 54 grams respectively, can also hardly be classed as showing important degrees of brain atrophy (see below).

14. One case (VII, 11.31) must be excluded from the present analysis, because total brain sections are in process of making (case of syringomyelia).

15. There remains a group of 12 cases, excluding I, XIV, V from the original 19, *i.e.* I as general paretic, XIV as arteriosclerotic dement, V as epileptic, IV, XV and XIII as having atrophic brains, VI as syringomyelia (analysis unfinished). We accordingly remain with one normal-looking brain in twelve.

16. The residue of normal-looking brains, with the above eight omissions, consists of the following twelve:—

Case.	Sex.	Age.	Onset.	Duration.	Diagnosis.
2 (13.44)	F.	44	44	1½ mos.	Central neuritis.
3 (13.6)	M.	44	43	2½ mos.	General paresis? Korsakoff.
8 (12.37)	F.	71	71	8 mos.	Senile psychosis.
9 (13.16)	M.	77	75	22 mos.	Senile psychosis; cerebral arteriosclerosis.
11 (12.7)	M.	43	42	3 mos.	Exhaustion psychosis.
12 (13.41)	F.	60	50	10 2/3 yrs.	Unclassified (paranoia).
14 (12.11)	M.	84	67	14 yrs.	Involution-melancholia.
16 (11.36)	F.	30	29	20 mos.	Manic-depressive psychosis.
17 (13.29)	F.	53	41	13½ yrs.	Manic-depressive psychosis.
18 (13.7)	F.	64	20	2÷1½ yrs.	Unclassified manic depressive.
19 (10.9)	F.	56	42	14 yrs.	Paranoia or dementia precox.
20 (12.47)	F.	27	25	28 yrs.	Dementia precox (catatonia).

17. Attention is first directed to four cases of mental disease over ten years in duration; these are XIX, XIV, XII and XVII.

18. This group of cases in which gross registration of lesions might have been expected, was subjected to orienting microscopic examination:—

XIX, 10.9, shows strikingly few evidences of cell loss, but careful search discovered foci of cell loss in the right second temporal gyrus. This case, though of slow evolution and diagnosticated paranoia, is thought to have had hallucinations of hearing as well as of sight. The delusions were largely of jealousy and otherwise sexual. One attack of so-called "cerebral congestion" at 40.

XIV, 12.11, involution-melancholia, 84 years at death, exhibited considerable cell loss in outer layers without marked satellitosis. Marked cell loss in calcarine region.

XII. 12.41, unclassified paranoic case, died at 60, showed fairly numerous cell losses.

XVII, 13.29, manic-depressive psychosis, died at 53, showed numerous cell losses, especially in upper layers.

19. According to a principle mentioned in the Worcester analysis, it would be unlikely that induration should register itself in brains undergoing gliosis in less than three months. There were three cases (II, III, XI), of which II was the case of possible central neuritis with marked acute cell changes ample to explain roughly the brief mental disease, III showed numerous acute cell changes probably quite consistent with the mental picture (Korsakoff's psychosis) and XI showed cell losses, perhaps of long standing

(although there were overt symptoms for three months only) together with acute cell changes.

20. The group of intermediate duration, three months to three years, comprises 5 cases, XVI, VIII, XX, XVIII, IX. Of these, VIII, age 71, and IX, age 77, attract attention on the score of age; both showed cell losses, in the former focal with perivascular gliosis, in the latter marked diffuse losses. Of the three remaining, two are manic-depressive cases (XVI, 11.36, and XVIII, 13.7) and one (XX) catatonic dementia precox. All three showed moderate degrees of cell loss.

21. Accordingly, it is plain that the search for functional psychoses which shall be above all neuropathological reproach is an exceedingly elusive task and possibly never to be rewarded. In a forthcoming communication we shall deal with the detailed microscopic picture in five of the cases of this series (XII, XVI, XVIII, XIX, XX) since these five appear to be the least likely of all our series of 153 cases to show important microscopic lesions.

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XLVII

AN ANATOMICAL SEARCH FOR IDIOPATHIC EPILEPSY.

Being a First Note on Idiopathic Epilepsy at Monson State Hospital, Massachusetts,* U.S.A.

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General Method of this Study.

The logical method of this study was borrowed from similar studies in the field of insanity.[1] These studies, carried on in the pathological service of the State Board of Insanity of Massachusetts,[2] had as their object an anatomical approach to

* Being a contribution from the *State Board of Insanity*, whole number, 46 (1915, 12). The previous contribution was 1915, 11, by E. E. Southard and M. M. Canavan, entitled "Notes on the Relation of Somatic (Non-Neural) Neoplasms to Mental Disease," published in the *Interstate Medical Journal*, Vol. xxii., No. 7, pp. 738-751, July 1915.

the problem of the functional psychoses, so-called. In the field of epilepsy a similar study confronts the idiopathic group, so-called. The same anatomical approach to the problem of feeble-mindedness, notably of the higher grades, "where little or nothing can be found in the brain," should be made as soon as possible.[3] It is conceivable, too, that the method might be applied to criminal anthropology, in so far as it studies somatic anomalies.[4]

The method consists in dividing one's available anatomical material, so far as this consists of clinically well-described cases, into two groups, group A to consist of cases with well-marked coarse brain lesions, and group B to consist of cases without such lesions. If the problem were the usual one of matching up various clinical and anatomical findings in group A, we should find ourselves adopting the approved and classical methods of the localising neurologist; and we find ourselves with two sub-groups of cases—A1 in which the matching of symptoms and lesions seems successful, and A2 in which the matching has failed. But in dealing with group B (the normal-looking brain group), our refinements proceed in the opposite sense, for we are endeavouring to find cases in which it is both practically and theoretically impossible to do any matching of symptoms and lesions, just because, by hypothesis, there are no lesions. The further analysis of group B shows sub-group B1, in which, perhaps, a subsequent careful dissection or microscopic examination reveals suspicious conditions that *might* have had to do with the symptoms, leaving us with sub-groups B2, in which careful dissection and available microscopic methods fail to show anything at all adequate to explain the symptoms.

Groups A1 and B2, then, form the extreme members of the familiar threefold statistical division, leaving a group composed of A2+B1, which are insusceptible to present-day research methods.

Group B2, then, is a refined group of cases with normal-looking brains, in which a suitable group of *idiopathic* cases of *epilepsy* (as in the present study), or of *functional psychoses*, or of *feeble-minded having good brains that do not work well*, or of *environmental delinquents* may be sought. All the while, it goes without saying, one does not deny that many of the cases in group B1 or group A2, or even group A1, may be equally *idiopathic*, or *functional*, or *non-pragmatic*, or *environmental*. We

simply choose not to use these groups in working out the problem of "discords played on good instruments."

Is epilepsy (or, better, are certain forms of epilepsy) ever a *discord* played (*e.g.*, by some unknown physico-chemical condition) *on a good instrument* (*i.e.*, a brain destitute of anomalies, lesions, or other conditions of an epileptogenic nature)? What, then, is the further special nature of such cases?

That the brain of a patient who has been subject to epileptic attacks, perhaps daily for a decade without remission, will appear normal at the autopsy table, seems at first sight rather unlikely.

Add to the epilepsy feeble-mindedness and dementia, as well as frequent maniacal attacks (which often precede and follow the convulsions), and the structural normality of the brain must seem still more dubious.

The clinical manifestations of the disease are so spectacular that one looks for somatic findings to correspond. Yet we are confronted with a rather large group of cases wherein the duration has extended over a period of years, convulsions being frequent and severe, associated with maniacal attacks and other active mental symptoms: as delusions, hallucinations, &c., where the brain reveals little or nothing at autopsy to account for these various symptoms of mental disturbance.

The purpose of this note is (1) to present a series of more or less normal-looking brains and a series of abnormal-looking brains, with clinical data, and study these data comparatively to delimit the resulting problems; (2) to compare the results of this analysis with those obtained in the analysis of normal-looking brains in the sane; and (3) finally to take the residue of cases which, after careful study, appear to be truly idiopathic, both from a clinical and anatomical standpoint, and prepare a line of research which shall come still nearer to determining the genuineness of their idiopathic nature.

That we should be prepared to find few or no truly idiopathic cases of epilepsy is indicated by a frequently quoted generalisation of Allen Starr,[5] who, speaking of organic disease of the brain, says, "I believe that epilepsy is always an expression of such disease." Aldren Turner[6] goes almost as far, appealing likewise to "the inherited neuropathic predisposition revealed by well-marked stigmata of degeneration."

We are afraid, however, that some statements of this order

are only pious wishes. Some argue that epilepsy *must* be "organic," as others argue that insanity *must* be due to brain disease. The phenomena are so striking that these disputants feel that the burden of proof is on the man who argues against this generalisation.

Brilliant observations of an anatomical or surgical nature, as well as ingenious modern work in histology, fall far short of the 100 per cent. structural correlation which is the darling wish of numerous epileptologists. The work of Meynert,[7] Chaslin,[8] Clark and Prout,[9] Onuf,[10] John Turner,[11] L. W. Weber,[12] Alzheimer,[13] to mention only a few of the workers, as well as some work by Worcester,[14] and later by one[15] of the present writers, is all subject to the charge of dealing with too small groups or the most favourable cases. At any rate, we believe that the anatomical approach to idiopathic epilepsy has been neglected. If the structurality ("organic nature") of epilepsy is entirely a histological matter in any group of cases, it will be important to study such a group intensively. Could it be that the pertinent "changes" are "reversible" in a chemical or in some more general logical sense? Are the brains in the meantime between convulsions quite normal?

Our denominator consists of 205 random cases of clinically certain epilepsy, which came to autopsy during the years 1901 to 1914. The autopsies were performed by various hands, largely by Drs A. E. Taft, M. B. Hodskins, L. B. Alford, and D. A. Thom. The standards have varied somewhat, and a few cases in the series have had to be excluded for extraneous reasons.

The group is, however, entirely representative and unselected, and is, beyond question, a fair sample of institutional epilepsy drawn from all parts of Massachusetts.

We found 129 instances of definitely abnormal-looking brains —brains of such an appearance that their possessors could not be safely taken to have idiopathic epilepsy. By this statement we do not mean that the abnormalities seen were the causes or effects of the epilepsy, or necessarily correlatable with the epilepsy. We regard them, however, as unsuitable for any more particular or intimate analysis of functional or idiopathic cases, simply because the lesions or anomalies found might be related with the epilepsy.

The residue of seventy-six cases shows normal-looking brains

—without marked chronic leptomeningitis or visible lesions of the brain substance, either acute or chronic.

The brains were not subject to extensive dissection at the autopsy table, as, for example, the majority of psychopathic subjects examined by Dr S. C. Fuller at Westborough [16]; by consequence it may be surmised that 37·1 per cent. (76 : 205) is rather too high a percentage of normal-looking brains, but it is evident that it is among these seventy-six that we must look for cases that shall be idiopathic epilepsy above reproach.

Perhaps the first point lodges in the question of dementia and feeble-mindedness.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
With acquired dementia -	35	...	46	...
With feeble-mindedness -	29	90	69	90
Without mental symptoms alone - - - - -	4	...	1	...

Thus we find thirty-five normal-looking brains in which we must explain not merely the absence of lesions which might be correlated with the epilepsy, but also which might be related with dementia, and we must take into consideration that twenty-nine were regarded as feeble-minded to start with. We accordingly obtain a small residue of twelve cases which had normal-looking brains, but were neither feeble-minded nor demented. This latter group is the most promising for a study of truly idiopathic cases. The situation at this point presents two problems: Firstly, How can thirty-five cases of epileptic dementia be explained if the brains are not diseased? Secondly, Does the group of twelve cases without feeble-mindedness or dementia represent the problem of functional epilepsy?

Leaving for the moment the problem of dementia and normal-looking brains (perhaps more truly a psychiatric problem) let us consider the possibly idiopathic group. A superficial analysis shows four cases of this group with, not, to be sure, feeble-mindedness or dementia, but certain mental symptoms of an acute

nature, delusions, hallucinations, maniacal states, &c. It is clear that, however important these cases may be, they do not represent faithfully the idiopathic group of pure epilepsy.

We find ourselves, therefore, with a residue of eight cases without feeble-mindedness, dementia, or other psychotic symptoms. Four of these six cases must, however, be still further questioned before they gain admission to that group best suited for the study of idiopathic epilepsy. Two of these cases presented a mild chronic leptomeningitis of unknown duration. To prove an association between this condition and epilepsy would require special research; yet to relieve all doubt and suspicion in the mind of the most exacting critics, both cases may be, with regret, discarded. Another case (♯699,04—4) had a paralysis of the left side of the face without any other symptoms organic in nature. She has been retained in the group with this explanation.

Case (♯315,08—24).—The description of the convulsions in patient's record is rather suggestive of organic epilepsy. But otherwise the case meets all requirements of idiopathic epilepsy, both clinically and pathologically, and may be retained for further study.

Tables are here introduced giving some general and special facts covering these groups, here briefly termed "normal" and "abnormal."

IV.—Tabulation of Findings in Abnormal and Normal-Looking Brain Series.

The following tables give the general results of the inquiry :—

Table I.

Autopsied cases - - - -	205
Brains, abnormal (substantial lesions) -	129
Brains, normal (no gross substantial lesions) -	76

76 Normal-Looking Brains.

Males, 41. Females, 35.

Normal-looking brains and coverings - -	68
Normal-looking brains with leptomeningitis -	8

Brains and Coverings Normal Looking—

Without mental symptoms	6
With dementia	30
With feeble-mindedness	28
With active mental symptoms *	4
	68

Normal-Looking Brains, but with Leptomeningitis—

Without mental symptoms	2
With dementia	5
With feeble-mindedness	1
With active mental symptoms	...
	8

129 Abnormal Brains.

Males, 65. Females, 64.

Abnormal Brains with Normal Coverings—

Without mental symptoms	12
With dementia	37
With feeble-mindedness	60
With active mental symptoms *	1
	110

Abnormal Brains and Coverings—

Without mental symptoms	1
With dementia	9
With feeble-mindedness	9
With active mental symptoms (alone)	...
	19

Table II.—Age at Onset.

	Normal.	Abnormal.
Males	41	65
Females	35	64
	Average age of onset, 18·7 years.	Average age of onset, 19·1 years.
Total	76	129
Age at onset	72 cases. 4 unknown.	118 cases. 11 unknown.

* With active mental symptoms and also feeble-mindedness or dementia, seventeen cases.

TABLE II.—*continued.*

Age at Onset.	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Under 1 year - -	4	5·5	13	11·0
From 1 to 3 years -	12	16·6	23	19·4
„ 4 „ 5 „ -	4	5·5	5	4·2
„ 6 „ 10 „ -	4	5·5	18	15·2
„ 11 „ 15 „ -	18	25·0	11	9·3
„ 16 „ 20 „ -	9	12·5	7	5·9
„ 21 „ 30 „ -	5	6·9	11	9·3
„ 31 „ 40 „ -	5	6·9	9	7·6
„ 41 „ 50 „ -	6	8·3	10	8·3
„ 51 „ 60 „ -	2	2·7	5	4·2
„ 61 „ 70 „ -	2	2·7	3	2·5
„ 71 „ 80 „ -	1	1·3	3	2·5

TABLE III.—DURATION FROM ONSET.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Under 2 years - -	...	...	...	...
From 2 to 4 years -	4	5·5	2	1·7
„ 5 „ 9 „ -	5	6·9	18	15·2
„ 10 „ 14 „ -	16	22·2	20	17·0
„ 15 „ 19 „ -	12	16·6	17	14·4
„ 20 „ 24 „ -	16	22·2	12	10·1
„ 25 „ 29 „ -	9	12·5	15	12·7
„ 30 „ 34 „ -	6	8·3	12	10·1
„ 35 „ 39 „ -	...	...	3	2·5
„ 40 „ 44 „ -	...	...	8	6·8
„ 45 „ 49 „ -	2	2·7	5	4·2
„ 50 „ 54 „ -	...	...	4	3·3
„ 55 „ 59 „ -	1	1·3	1	·8
„ 60 „ 64 „ -	1	1·3	1	·8
Unknown - - -	4	...	11	...

TABLE IV.—AGE AT DEATH.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Between 10 and 20 years	9	11·8	21	16·2
„ 21 „ 30 „	24	31·5	26	20·1
„ 31 „ 40 „	11	14·4	17	13·1
„ 41 „ 50 „	15	19·7	15	11·7
„ 51 „ 60 „	5	6·6	29	22·4
„ 61 „ 70 „	7	9·2	11	8·5
„ 71 „ 80 „	5	6·6	7	5·4
Over 80 years - -	...	...	3	2·3
Average age at death -	38·9 years		41·44 years	

TABLE V.—HEREDITY.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Same heredity - -	9	13·8	16	12
Allied heredity - -	7	10·6	10	8
Alcoholic hist. in pat. -	8	12·5	13	10
Syphilitic hist. in pat. -	1	1·5	3	2·3
Alcohol and same heredity - - -	1	...	2	...
Alcohol and allied heredity - - -	2	...	2	...

Here "Same" heredity means epilepsy. "Allied" means other psychopathic conditions.

Table VI.—Mental Status.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Demented - - -	35	46	46	35·6
Feeble-minded - -	29	38	69	53·5
Active mental symptoms -	4	5	1	1
Without mental symptoms	8	10	13	10
	76		129	
Active mental symptoms, with feeble-mindedness	21	...	4	...
Active mental symptoms, with dementia - -	23	...	14	...

Table VII.—Frequency of Convulsions.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
1 or less per month -	9	12	26	21·1
2 per month - -	3	4	9	7·3
3 to 5 per month -	19	25·7	28	22·7
6 „ 10 „ -	13	17·5	22	17·8
11 „ 15 „ -	11	15	13	10·5
16 „ 20 „ -	6	8·1	9	7·3
21 „ 25 „ -	2	2·5	4	3·2
26 „ 30 „ -	4	5·4	5	4
31 „ 35 „ -	3	4	3	2·4
36 „ 40 „ -	3	4	1	·8
Over 40 „ -	1	1·3	3	2·4

TABLE VIII.—ASSIGNED CAUSES OF EPILEPSY.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Head injuries	7	9·1	13	10·0
Alcohol	4	5·2	2	1·5
Encephalitis	1	1·3	3	2·3
Scarlet fever	3	3·9	2	1·5
Teething	3	3·9	3	2·3
Menopause	2	...	1	·7
Sunstroke	...	2·6	...	...
Meningitis	...	...	4	3·1
Hereditary syphilis	...	...	...	...
Acquired syphilis	...	...	1	·7
Cerebral hæmorrhage	...	...	1	·7
Chorea	...	...	1	·7
Indigestion	...	...	1	·7
Measles	...	...	...	...
Emotion, fright, grief	2	2·6	4	3·1
Overwork	1	1·3	3	2·3
Congenital	...	...	6	4·6
Rachitis	...	...	1	·7
Growth on frontal bone	1	1·3	...	...
Unknown	52	68·4	83	64·3

TABLE IX.—CAUSES OF DEATH.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Pulmonary tuberculosis	9	11·7	12	10
Lobar pneumonia	2	...	7	...
Broncho-pneumonia	6	...	10	...
Pulmonary œdema	4	...	14	...
Pulmonary thrombi	...	...	1	...
Pulmonary abscess	...	...	1	...
Pulmonary apoplexy	...	...	1	...

TABLE IX.—*continued.*

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Asphyxia	3	...	4	...
Chronic myocarditis	2	...	3	...
Chronic endocarditis	4	...	4	...
Acute dilatation of heart	3	...	2	...
Cardiac paralysis	1	...	1	...
Internal hæmorrhage	...	...	1	...
Cancer of liver	1	...	...	...
Chronic pericarditis	...	...	1	...
Chronic internal nephritis	...	...	1	...
Cirrhosis of liver	...	...	1	...
Diabetes mellitus	...	...	1	...
Peritonitis, acute	...	...	1	...
Peritonitis, T.B.	...	...	1	...
Septicæmia	...	...	2	...
Acute dilatation of stomach	...	...	1	...
Acute gastro-enteritis	1	...	1	...
Scald	...	...	1	...
Drowning	...	...	1	...
Cancer of prostate	...	...	1	...
Meningitis, T.B.	1	...	...	...
Meningitis, cerebro-spinal	...	...	1	...
General paresis	...	...	1	...
Amytrophic lateral sclerosis	...	...	1	...
Cerebral hæmorrhage	1	...	8	...
Cyst of brain	...	...	2	...
Softening of brain	...	...	2	...
Abscess of brain	...	...	1	...
Tumour of brain	...	...	1	...
Anæmia of brain	...	...	1	...
Intestinal paralysis	1	...	...	...
Epilepsy	35	...	37	...
Gangrene	1	...	1	...
Carcinoma	1	...	...	...

TABLE X.—CORRELATION OF ALCOHOL AND EPILEPSY IN RELATION TO AGE AT ONSET.

	Normal.		Abnormal.	
	Number of Cases.	Per Cent.	Number of Cases.	Per Cent.
Congenital - - -	3	23	...	...
Between 1st and 2nd year	1	7·7	1	4·5
„ 2nd „ 3rd „	2	15·4	3	13·7
„ 3rd „ 4th „	1	7·7	2	9·1
„ 4th „ 5th „	...	...	2	9·1
„ 5th „ 6th „	1	7·7	...	...
„ 6th „ 10th „	...	...	1	4·5
„ 11th „ 15th „	2	15·4	4	18·2
„ 16th „ 20th „	2	15·4	2	9·1
„ 21st „ 25th „	...	...	...	...
„ 26th „ 30th „	1	15·4	1	4·5
„ 31st „ 35th „	...	...	3	13·7
„ 36th „ 40th „	...	...	...	...
„ 41st „ 45th „	...	...	1	4·5
„ 46th „ 50th „	...	...	2	9·1

The groups of Table I. correspond to those mentioned in the first section of this note as follows:—

Group A, composed of 129 abnormal-looking brains.

Group A1, composed of 19 brains, as it were doubly abnormal, since both the brain substance and the nutrient membrane are diseased.

Group A2, composed of 110 cases with substantial lesions, not here further analysed. These two groups are without doubt very heterogeneous, but do not contribute readily to the idiopathic problem. Further study might throw many of group A2 into A1.

Group B, composed of 76 normal-looking brains, *i.e.*, without substantial lesions.

Group B1, composed of 8 brains substantially normal-looking but with chronic leptomeningitis, the significance of which lesion is questionable.

Group B2, composed of 68 brains substantially normal-looking and without chronic pial changes.

The following table shows our best approximation to an idiopathic group of sane epileptics, and is obtained by subtracting

from groups B1 and B2 all cases with feeble-mindedness, dementia, or acute psychotic symptoms.

The remaining 6 cases, already clinically idiopathic, must be further studied microscopically to prove that they are histologically intact. This work will be done and reported at a later date. In the summary of the present paper will be embodied some general and special facts covering a study of a comparative data between what is briefly termed normal and abnormal brains.

Summary and conclusions:—

1. Seventy-six of 205 brains of institutional, but otherwise unselected, epileptic subjects, *i.e.*, 37 per cent., yielded brains without substantial lesions visible to the naked eye upon superficial examination or dissection.

2. This percentage of "normal-looking" brains is rather higher than has hitherto been found in institutional, psychopathic, *non*-epileptic subjects, although the dissections in the epileptic group have probably not been so extensive as in the psychopathic group.

3. A study has been made of 76 epileptics with normal-looking brains, with the hope of securing a number of "idiopathic" cases for special examination.

4. In order to secure a group of pure epilepsy, 68 cases had to be excluded as being complicated with feeble-mindedness, acquired dementia, or other psychotic symptoms, leaving 8 apparently non-psychotic epileptics for study. Of these 8, 1 had facial palsy, 1 had organic-looking symptoms, and 2 had chronic leptomeningitis. Dismissing the 2 cases of chronic leptomeningitis, we have 6 cases from which a truly idiopathic brain, from a histological point of view, may be isolated, and it is upon these 6 brains that further study must be made.

5. The whole series affords an opportunity for general conclusions on certain classical questions of epileptology, *e.g.*:—

Age at Onset, Table II.—(*a*) Seventy-two cases out of a total of 76 with normal-looking brains where the age at time of first convulsions was known. *Eighteen* (25 *per cent.*) *began between eleven and fifteen years*, a period quite significant for the disturbance of the nervous system, already predisposed to psychochemical changes. *Of the* 118 *cases with abnormal brains, only* 9·3 *per cent. had their onset during this same period.* (*b*) The abnormal series show that the percentage of cases (11 per cent.) where the age at onset was under one year was twice as high

(5·5 per cent.) as the normal series (suggesting birth injuries and congenital defects). All those cases where the epilepsy began after the fortieth year were about equally divided between the normal and abnormal group.

Duration of Epilepsy, Table III.—The cases where the duration was of thirty-five years or more were divided as follows:—18·4 per cent. abnormal group; 5·3 per cent. normal group. Those with shorter durations were about equally divided between the two groups.

Age at Death, Table IV.—Average age of patient at time of death, in normal group, 38·9 years; abnormal group, 41·44 years.

Heredity, Table V.—Heredity present in 24 per cent. normal cases; 20 per cent. abnormal cases, being about equally divided in either group into the same and allied types of heredity.

Mental Status, Table VI.—Only 10 per cent. of the cases in either group that did not present mental symptoms, dementia being more frequent in the normal group (46 per cent.), while feeble-mindedness predominated in abnormal group (53 per cent.).

Number of Convulsions, Table VII.—*Cases with minimum number of convulsions,* one or less a month, *belonged largely to abnormal series,* while the *cases where the convulsions occurred* once a day or more *frequently* were *usually found in the normal series.*

Assigned Causes of Epilepsy, Table VIII.—The assigned causes varied so widely, and in so many instances were unknown, that the data were of little significance, excepting that head injuries was given as the cause in 9·1 per cent. in the normal series and in 10 per cent. in the abnormal series. Alcohol, normal series, 5·2 per cent.; abnormal series, 1 per cent. The causes of death were also so numerous that the data are of little importance, excepting that tuberculosis was the cause of death in about 10 per cent. of all cases in either group.

Alcohol and Syphilis in Patients, Table IX.—Alcohol, 12·5 per cent. normal group; 10 per cent. abnormal group. Syphilis, 1·5 per cent. normal group; 2·3 per cent. abnormal group.

We feel that, contrary to the expression of the numerous authors already quoted, there still remains some doubt that all epilepsies are organic in nature, and it has been the purpose of this note to introduce a more logical method of anatomical search for idiopathic epilepsy than has hitherto been applied to the problem.

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XLVIII

DATA CONCERNING DELUSIONS OF PERSONALITY WITH NOTE ON THE ASSOCIATION OF BRIGHT'S DISEASE AND UNPLEASANT DELUSIONS.*

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Abstract

Previous work on somatic delusions.
Suggestion that allopsychic delusions are as a rule in some sense autopsychic.
A genetic hint from general paresis (frontal site of lesions in cases with autopsychic trend.)
Mental symptomatology of general paresis.
Work on fifth-decade psychoses.
Statistical summary.
Group with pleasant (or not unpleasant) delusions.
Three cases of senile dementia, delusions of grandeur, and frontal lobe changes.
Three cases with religious delusions.
Remainder of pleasant-delusion group.
Group with unpleasant delusions.
Nephrogenic (?) group.

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THE suggestions here put forward concerning personal (autopsychic) delusions are based on material of the same sort as that previously analyzed for a study of somatic and of environmental (allopsychic) delusions. Our conclusions are also influenced by two analyses of the types of delusion found in general paresis. Moreover, at a period subsequent to the analysis presented here, some work on fifth-decade insanities had been completed, and the delusional features constantly found in the functional cases of insanity developing at the climacteric, entered to modify our general point of view.

The situation may be summed up as follows:

The accessibility to analysis of the clinical and anatomical data at the Danvers State Hospital was such as to prompt the use of its card catalogues for statistical work upon delusions. The more so, because in a period of enthusiasm over the Wernickean trilogy (autopsyche, allopsyche, somatopsyche) of conscious phenomena, the Danvers catalogue had attempted to divide the delusions recorded into the three Wernickean groups. Putting these clinical data side by side with the anatomical data, we were speedily able to single out those cases with normal or normal-looking brains and thus to secure a group approximately composed of functional cases of insanity.

It shortly developed, as to the *content* of delusions, that somatic delusions were exceedingly prone to parallel the conditions found in the trunk-viscera and other non-nervous tissues of the subjects at autopsy.[1] A subsequent study has confirmed this conclusion for the distressing hypochondriacal delusions found in climacteric insanities, which delusions, however distressing, are often far less so than the true conditions found at autopsy. And it may be generally stated that the clinician can get very valuable points concerning the somatic interiors of his patients by reasoning back from the contents of their somatic delusions.

But how far can we, as psychiatrists, reason back from the contents of environmental delusions, e. g. those of persecution, to the actual conditions of a given patient's

environment? In a few cases it seemed that something like a close correlation did exist between such allopsychci delusions and the conditions which had surrounded the patient—the delusory fears of insane merchants ran on commercial ruin, and certain women dealt in their delusions largely with domestic *debâcles*. But on the whole, we could *not* say that, as the somatic delusions seemed to grow out of and somewhat fairly represent the conditions of the soma, so the environmental delusions would appear to grow out of or fairly represent the environment.

Thus, however brilliant an idea was Wernicke's in constructing the *allopsyche* (or, as it were, social and environmental side of the mind) for the purpose of classification, our own analysis promised to show that for genetic purposes the allopsyche was much less valuable. These delusions having a social content pointed far more often inwards at the personality of the patient than outwards at the conditions of the world. And case after case, having apparently an almost pure display of environmental delusions, turned out to possess most obvious defects of intellect or of temperament which would forbid their owners to react properly to the most favourable of environments. Hence, we believe, it may be generally stated that the clinician is far less likely to get valuable points as to the social exteriors of his patients from the contents of their social delusions than he proved to be able to get when reasoning from somatic delusions to somatic interiors. Put briefly, the deluded patient is more apt to divine correctly the diseases of his body than his devilments by society.

Our statistical analysis, therefore, set us drifting toward disorder of personality as the source of many delusions apparently derived *ab extra* and tended to swell the group of autopsychic cases at the expense of the allopsychic group,

In the statistical analysis of a group of cases corresponding roughly with the so-called functional group of diseases. we find false beliefs about the soma on a somewhat different plane from those about the patient's self and his worldly fortunes. We can even discern through the ruins of the paretic's reaction that his false beliefs concerning the body are often not so false after all, and that his damaged brain

of itself is not so apt to return false ideas about his somatic interior as about his worldly importance and plight. There then seems to be more reality about somatic than about personal delusions: the contents of somatic delusions are rather more apt to correspond with demonstrable realities than the contents of personal delusions. Accordingly our analysis of delusional contents includes a hint also as to genesis. Taken naïvely, the facts suggest a somatic genesis for somatic delusions exactly in proportion as these delusions are not so much false beliefs as partially true ones.

What genetic hint have we for the delusions concerning personality? One genetic hint was obtained from a correlation of delusions with lesions in general paresis,[2] in which disease perhaps the most profound and disastrous of all alterations of personality are found. Amidst the other alterations of personality found in paresis, autopsychic delusions are characteristic: indeed allopsychic delusions are conspicuously few in our series. And, as above, the somatic delusions, fewer in number, can be fairly easily correlated with somatic lesions, or else with lesions of the receptor apparatus (thalamus) of the brain.

Now it was precisely the cases with autopsychic delusions, as well as with profound disorder of personality in general, that showed the brunt of the destructive paretic process in the frontal region. The other not-so-autopsychic cases did not show this frontal brunt, but were less markedly diseased at death and had a more diffuse process.

Our genetic hint from paresis, therefore, inclines us to the conception that this disorder of the believing process is more frontal than parietal, more of the anterior association area than of the posterior association area of the brain. And if we can trust our intuitions so far, the perverted believing process is thus more a motor than a sensory process, more a disorder of expression than a disorder of impression, more a perversion of the *will to believe* than a matter of the rationality of a particular *credo*.

Again we may appear to burst through from an undergrowth of statistics into the clear field of truism. False beliefs are more practical than theoretical, more a matter of practical conduct than of passive experience, more a change

of reagent than a reaction to change. The man on the street or even many a leading neurologist would perhaps accept this formula as his own.

Certainly in general the least satisfactory of these chapters on the nature of delusions was the chapter on environmental effects,[3] and this perhaps because the results seemed so nearly negative.

A further contribution to delusions of environmental nature was somewhat unexpectedly derived from a piece of work on the general mental symptomatology of general paresis.[4] Dichotomizing the paretics (all autopsied cases) into a group with substantial, i. e., encephalitic, atrophic or sclerotic lesions of the cortex and a group without such gross lesions or else with merely a leptomeningitis, I found the latter (or anatomically mild) group to be characterized by a set of symptoms which were all "contra-environmental," whereas the former (or anatomically severe) did not thus run counter to the environment. The conclusions of that paper, so far as they concern us now, are as follows:—

The "mild" cases showed a group of symptoms which might be termed *contra-environmental*, viz. *allopsychic delusions*, *sicchasia* (refusal of food), *resistiveness*, *violence*, *destructiveness*.

The "severe" cases showed a group of symptoms of a quite different order, affecting *personality* either to a ruin of its mechanisms in *confusion* and *incoherence*, or to mental quietus involved in *euphoria*, *exaltation*, or *expansiveness*.

The most positive results of this orienting study appear to be the unlikelihood of euphoria and allied symptoms in the "mild" or non-atrophic cases and the unlikelihood of certain symptoms, here termed *contra-environmental*, in the severe or atrophic cases. Perhaps these statistical facts may lay a foundation for a study of the pathogenesis of these symptoms. Meantime the pathogenesis of such symptoms as amnesia and dementia cannot be said to be nearer a structural resolution, as these symptoms appear to be approximately as common in the "mild" as in the "severe" groups.

But in both papers dealing with paresis [2, 4] we rest under the suspicion that the delusions are possibly of cerebral

manufacture. Of course, a lesion somewhere outside the brain is not unlikely to be projected through the diseased brain, and *somatic* delusions in the paretic are rather likely to represent something in the viscera.

It was desirable to get back to normal-brain material, to learn how the *intrinsically normal* brain[5] could perhaps produce delusions from a particular environment. Could a particularly "bad" environment actually *produce* delusions?

By chance, at about this stage in our studies of delusions, some work on fifth-decade insanities[6] was completed. This work seemed to show that the most characteristic (non-coarsely-organic) cases of involutional origin were much given to delusions (each of 24 cases studied), somewhat more so than to the hypochondria and melancholia which we commonly ascribe to the involution period. But this result is equivocal as to the environmental (i. e. allopsychogenic) power to produce delusions, since one could not rid oneself of the suspicion that the delusions were due to the degenerating brain.

To return to our former results with the normal-looking brain:

Case after case of the quasi-environmental group proved to be more essentially personal than environmental, until at last it almost seemed that the environment could seldom be blamed for any important share in the process of false belief. In short, we seemed to show that environment is seldom responsible for the delusions of the insane.

Be that as it may, we secured several lines of attack on the delusions of personality by our study of quasi-environmental delusions. First, we were irresistibly led to a consideration of the emotional (pleasant or unpleasant) character of the delusions. We heaped up a large number of unpleasant delusions in that (quasi-environmental, but actually) personal group. It is interesting to inquire, accordingly, whether our more obviously autopsychic cases will also be possessed of an unpleasant tone. Secondly, we came upon the curious fact that cardiac and various subdiaphragmatic diseases were correlated with unpleasant emotion as expressed in the delusions. It was therefore important to inquire whether similar conditions prevailed in the new group.

Thirdly, we found ourselves inquiring whether our patients were victims of what might be termed a spreading inwards of the delusions (egocentripetal) or a spreading outwards thereof (egocentrifugal delusions). But this difference in trend, clear as it often is from the patient's point of view, remains to be defined from the outsider's point of view.

Again, it remains to determine, if possible, how far delusions are dominated respectively by the intellect or the emotions, or even by the volitions.

As before, I begin with a brief statistical analysis.

Summary

Danvers autopsy series, unselected cases	1000
Cases with little or no gross brain disease	306
Cases listed as having autopsychic delusions	106
Cases listed as having only autopsychic delusions	50
Cases for various reasons improperly classified	13
Cases of general paresis in which gross brain lesions were not observed	15
Residue of autopsychic cases	22

The group of 22 cases thus sifted out can be studied from many points of view. We may recall that our former study of allopsychic delusions proved that a large proportion of delusions concerning the environment were in all probability not essentially derived from the environment. Their contents might relate to the environment, but their genesis could better be regarded as autopsychic (intrapersonal). In fact we really found only 6 out of 58 cases of pure allopsychic delusions, which could be safely taken as showing so much coincidence between anamnesis and delusions that a correlation could be risked.

Following the method of our former work on somatic and on environmental delusions, we sought in the first instance *pure* cases of autopsychic delusion-information. For a variety of reasons, more than half of the original list, namely, 28 cases, had to be excluded. Many of these exclusions were due to the strong suspicion that the cases were really cases of general paresis, despite the normality

of the brains in the gross. The residue of 22 cases include, we are confident, no *instance of exudative* disease of the syphilitic group, though general syphilization cannot safely be ruled out in all cases.

There are two groups of cases, a group of eleven cases with delusions of a generally pleasant or not unpleasant character (in which group there is a small sub-group of three cases of octogenarians with expansive delusions reminding one of those of general paresis) and a group of eleven cases with delusions of an unpleasant character.

1. Cases Having Delusions of a Nature Pleasing or not Unpleasing to the Believer

The true emotional nature of the beliefs placed in this group cannot fairly be stated to be *pleasurable*. But, if not pleasurable, they may perhaps be stated to be *complacent*, *expansive*, or of *air-castle type*. The criteria of their choice have been largely negative: the patients are not recorded as expressing beliefs of a painful or displeasing character: in the absence of which we may suppose the beliefs to be either indifferent or actually pleasing in character.

Of the 11 cases whose delusions were supposedly of an agreeable nature or at least predominantly not unpleasant, there were 3 with delusions reminding one of general paresis. The ages of these three were 80, 84, and 87 respectively. They did not show any pathognomonic sign (e.g. plasma cells) of general paresis. They all showed in common very marked lesions of the cortex, including the frontal regions (in two instances the extent of the frontal lesions was presaged by focal overlying pial changes) .999 was a case of pseudoleukemia with marked cortical devastation but without brain foci of lymphoid cells. Two of the cases showed cell-losses more marked in suprastellate layers; in the third there was universal nerve cell destruction, with active satellitosis caught in process.

Condensed notes concerning the cases with pseudoparetic delusions follow. Two of them, it will be noticed, yielded some delusions also of an unpleasant nature.

Case 1. (D. S. H. 10940, Path. 999) was a clever business man, Civil War veteran, who began to lose ground at 75 and died at 84. He was given during his disease to *boasting and perpetual writing about elaborate real estate schemes* and said he *owned a $100,000 concern* for the purpose.

The case was clinically unusual in that the picture of a *pseudoleukemia* was presented, with demonstration at autopsy of great

hyperplasia of retroperitoneal lymph nodes and grossly visible islands of lymphoid hyperplasia in liver and spleen. The brain weighed 1390 grams and showed little or no gross lesion, if we except a *pigmentation of the right prefrontal region* under an area of *old pias hemorrhage*. There was also a chronic leptomeningitis, with numerous streaks and flecks along the sulci, especially in the frontal region. There was little or no sclerosis visible in the secondary arterial branches and but few patches in the larger arteries. Microscopically the cortex proved to be far from normal: every area examined showed cell-loss, perhaps more markedly in the suprastellate layers than below.

Case 2. (D. S. H. 11980, Path. 1024) was a Civil War veteran who failed in the grocery business, was alcoholic, was finally reduced to keeping a boarding-house and grew gradually queer. Mental symptoms of a pronounced character are said to have begun at 75. Death at 80. Delusions reminded one of general paresis: worth *$5,000,000 a month, 108 years old, was to build a church: also, a woman was trying to poison him.*

Autopsy showed *caseous nodules in lung, coronary and generalized arteriosclerosis* (including moderate *basal cerebral*), mitral and aortic stenosis (the aortic valve also calcified). The *frontal pia mater* was greatly *thickened* and, although no gross lesions were noted in the cortex, the microscope brings out *marked* lesions in the shape of *cell losses* (especially in suprastellate layers) in all areas examined. There were no plasma cells in any area examined.

Case 3. (D. S. H. 12767, Path. 1185) was a widowed Irish woman, who died at 87. Previous history blank. *Extravagant* delusions of *wealth* were associated with a *fear of being killed.*

The autopsy showed little save chronic *myocarditis* with brown atrophy, *calcification* of part of *thyroid, non-united fracture of* neck of left *femur*, moderate *coronary arteriosclerosis.* The *brain* was abnormally *soft* (some of the larger intracortical vessels showed plugs of leucocytes possibly indicating an early encephalitis—Bacillus coli and a Gram-staining bacillus were cultivated from the cerebrospinal fluid.) Though the convolutions were neither flattened nor atrophied and absolutely no lesion was grossly visible, the cortex cerebri and also the cerebellum were found undergoing an active satellitosis with nerve-cell destruction in all areas examined.

The following three cases (IV, V, VI) present a certain identity from their delusions concerning messages from God (V thought he was God). It is very doubtful whether VI should be placed in the present group of Pleasant or Not Unpleasant Delusions, since the patient appears to have been "theomaniacal" as the French say, in a rather passive and unpleasant manner (God occasioned foolish actions:)

Placed on general statistical grounds at first in the Not Unpleasant group, Case VI should be transferred to the Unpleasant group. Case V's delusion (identification with God; expression of atonement?) was in any event episodic in a septicemia. Case IV ("happiest woman in the world"), was phthisical (cf. VII) Notes follow:

Case 4. (D. S. H. 4019, Path 218) Housewife, 37 years always cheerful, became *the happiest woman in the world, hearing God's voice* and being specially under God's direction. "Acute mania." Death from bilateral phthisis with numerous cavities and bilateral pleuritis. There were no other lesions except a small *sacral bed-sore*, a small *fibromyoma* of the uterine *fundus*, small slightly *cystic ovaries*, a slight *dural thickening*, and possibly a slight *general cerebral atrophy*. (wt. app. 1205 grams, marked emaciation.)

Case V. (D. S. H. 11742, Path. 852) was a victim of streptococcus septicemia (three weeks) who *said he was God.* Patient was a Protestant iron-worker of 59 years, who had lost an eye and had become unable to work about three months before death. *Aortic, cardiac, renal* lesions at autopsy. *Prostatic hypertrophy.* Dr. A. M. Barrett found *few changes in nerve cells*, except *fever changes.* One area in *left superior frontal gyrus* showed *superficial gliosis.*

Case VI. (D. S. H. 5345, Path. 867) was a "*primary delusional insanity,*" a salesman of 37 years, whose beliefs concerned *impressions direct from God,* in consequence of which he habitually knelt and prayed. Yet many of the actions which he felt he *must perform* were *foolish actions.* The patient died of *pneumococcus septicemia* during a lobar pneumonia. The *brain* showed a few *changes suggestive of fever* (A. M. Barrett). There were a few flecks of *atheroma in the aorta.* There was an acute *parenchymatous nephritis* with focal plasma cell infiltrations suggesting *acute interstitial nephritis.* This case appears to have shown one of the most nearly normal brains in the whole Danvers series.

The remainder of the Pleasant or Not Unpleasant Group as originally constituted consists of VII, a phthisical case (cf. IV); VIII, probably feeble-minded romancer, not deluded in the sense of self-deception (probably best excluded from present consideration); IX, probably not safely to be assigned to the Pleasant or Not Unpleasant Group, feeling passive in somewhat the same sense as Case VI (see above), suffering from auditory hallucinosis (superior temporal atellitosis, data of the late W. L. Worcester); X, delusion

of birth to superior station, possibly the object of mixed emotions, probably not pleasant; and XI, manic-depressive exaltation with grandiose utterances, long prior to death (if there had been lung tuberculosis at the basis of the ileac ulcers, it had long since healed).

Notes follow (VII-XI) and at the end a brief summary of the entire group (I-XI).

Case 7. (D. S. H. 8878, Path. 521) It is questionable whether the delusions classified in this case entitle it to inclusion in the present study. e.g. "*I was baptized in the Catholic Church* (patient a Protestant housewife) *with holy water, ink, and Florida water.*" Patient was variously designated, as "*dementia*" and as "*acute confusional insanity.*" Death in second attack at 26 (first attack at 22). Father also insane. Death due to *bilateral phthisis* with *tuberculosis of intestines and mesenteric glands, emaciation.* It is noteworthy that the brain weighed but 1038 grams. Dr. W. L. Worcester's microscopic examination showed acute nerve cell changes probably of the type of *axonal reactions.*

Case 8. (D. S. H. 8807, Path. 556) very probably a feeble-minded subject. At all events patient had done no work in his life, had been given to spells of restlessness and excitement, and had talked disconnectedly. Symptoms were thought to have dated from the tenth year. It is questionable whether a statement that he was *managing the Electric Railway and Shipbuilding Company* can be regarded as delusional, that is, as believed by the patient. Death was due to (perhaps septicemia from one abscess of jaw and to hypostatic penumonia), the brain appeared normal, but Dr. W. L. Worcester found, besides certain acute changes, also *satellitosis.* The question remains open whether the case should be regarded as defective or as belonging to the dementia praecox group.

Case 9. (D. S. H. 8605, Path. 568) had an ill-defined attack of mental disease and was in D. S. H. at 29. Thereafter, lived in Gloucester Almshouse, but at 51 became excited and was returned to D. S. H. where she died at 59. Possibly hallucinated: *someone called her mother* (single woman). Delusion: *the spirit is here* (Protestant). Patient was given to a stream of muttered, vulgar and incoherent talk. Possibly the case was residual from *hebephrenia.* Dr. W. L. Worcester found *cell changes in the superior temporal gyri* (finely granular stainable substance in practically all nerve cells) and *not* elsewhere. The correlation is suggestive with the probably auditory hallucinosis. The brain weighed 1190 grams. Death due to bronchopneumonia. Heart and kidneys normal.

Case 10. (D. S. H. 10145, Path. 928) a Danish fisherman, possibly manic-depressive, victim of three attacks at 40, 50, and

69 years. The first attack followed loss of wife, and delusions concerning being *born again* developed. The last attack showed few well-defined delusions, as patient was in a bewildered and incoherent state. One statement is characteristic: *if patient had remained in Denmark, he might have inherited the throne.* The autopsy showed most extensive *arteriosclerosis*, including *basal cerebral.* Death from general *anasarca* and *jaundice.* (cholelithiasis). There was some question of an acute *encephalitic lesion* in the tissues lining the posterior half of the *third ventricle.* Various *chronic* lesions (*splenitis, endocarditis, diffuse nephritis*), malnutrition.

Case 11. (D. S. H. 7767, Path. 792) was a case possibly of manic-depressive type (previous attacks Hartford Retreat and Danvers State Hospital) who worked as machinist between attacks and died at 70, having been in D. S. H. 8 years. Patient was greatly emaciated and anemic from chronic ulcers of ileum. There was also cholelithiasis. There was a mild *coronary atheroma* and slight mitral valve edge thickening.

The delusions expressed were those of *great wealth.* Patient also thought he was a *great poet.* No brain changes were found (A. M. Barrett).

Having attempted on the basis of certain statistical tags to constitute a group of cases having relatively normal brains and pleasant (or not unpleasant) delusions, we are forced to reconstruct our group upon viewing several cases more attentively.

Case VIII should be excluded as probably not delusional.

Case X might perhaps be transferred with propriety to the unpleasant-delusion group.

Certain cases of felt passivity under divine influence separate themselves out from the group; indeed VI and IX probably belong in the unpleasant-delusion group (see below).

These subtractions leave seven cases to deal with. Three of these seven, viz. I, II and III, are apparently best regarded as examples of frontal lobe atrophy, and their grandiosity may resemble that of certain cases of general paresis.

Of the remaining four, two, Cases IV and VII, are phthisical; one, Case VI, showed an episodic identification with God (incident in fatal septicemia), and one, Case XI, uttered manic-depressive exalted statements about wealth and poetical power.

I turn to a consideration of the unpleasant-delusion group, which as first constituted was to contain eleven cases (XII-XXII) but to which must be added three more (VI, IX, X).

Case XII should be at once excluded from present consideration on account of its microscopy.

Case 12. (D. S. H. 12282, Path. 942) died in a second attack of depression (manic-depressive insanity?). Catholic, always of a quiet and reserved disposition, happy in married life. Delusional attitude concerning an abortion which she said she had induced. "*Soul lost,*" "*I'll see hell.*"

Autopsy: Death from *gangrene of lung* and *acute fibrinous pericarditis. Erosion of cervix uteri. The edema of the brain, irregular pink mottlings of white substance*, and an exudative lesion of one focus in the pia mater of the right side suggested an *encephalitis* more marked on the right side. Microscopically a few small vessels showed plugs of polynuclear lencocytes. The nerve cells were affected by various acute changes. The visuo-psychic portion of an occipital section (right) showed suprastellate cell-losses of a somewhat focal character.

Of the remaining ten XIII-XXII), one, Case XIII is another of mixed emotions ("am Eve and have to suffer;" "in Purgatory;" etc) of a religious type. It is the only case in the unpleasant group with phthisis pulmonalis, (combined, however, with abdominal tuberculosis and nephritis).

Case 13. (D. S. H. 7361, Path. 499) was a somewhat defective Catholic woman (mother insane) always of a melancholy and reserved temperament. She had been ill-treated by husband, child had died, another had followed soon. She developed a belief that *she was Eve* and *had to suffer*. At hospital decided that *she was in purgatory* and expressed a variety of other religious beliefs. She also thought she was *ill-treated at hospital*. Her head was asymmetrical: skull thick and eburnated. Brain (1130 grams described as normal). *Chronic interstitial nephritis. Pulmonary and mesenteric tuberculosis.*

Of the remaining nine (XIV-XXII) all had grossly evident kidney lesions except two (XIV and XV). Of these two, XIV probably had renal arteriosclerosis and was in any case very gravely arteriosclerotic in general and suffered from cystitis. Case XV died apparently of starvation with

hepatic atrophy; it is a question whether "poverty" was or was not a delusion. Notes of XIV and XV follow:

Case 14. (D. S. H. 8741, Path. 500) was a German teacher, college-bred, of a reserved and melancholy turn of mind (mother insane). An attack at 39, another at 70. "*Both poor wife and son will starve.*" "*Perhaps they should be put out of reach of poverty,*" later felt he "*had caused death of wife and son on account of his expensive living.*" Autopsy: *chronic internal hydrocephalus, cerebral arteriosclerosis.* Brain weight 1180 grams. *Coronary sclerosis with calcification throughout, aortic and pulmonary valvular calcification hypertrophy of heart. Cystitis.*

Case 15. (D. S. H. 4454, Path. 237) was presumably a manic-depressive case, had in all four attacks, and died in the fourth attack (66 years). The day he arrived at the hospital, having not eaten for several days at the end of several months of *delusions of poverty* the case was called "acute melancholia,' and the cause of death assigned was starvation. The liver weighed 1102 grams and was fatty. There was a diffuse *thickening* and *clouding* of the *pia mater*, and the *dura* was firmly *adherent* everywhere to the skull.

Notes follow of seven cases (XVII-XXII) which show many lesions, are in a number of instances cardiorenal and in all instances renal. If it is permitted to count XIV also as renal, a list of eight cases out of the original list of eleven unpleasant-delusion cases is obtained in which nephritis of some type has been found. Case XIII, nephritis and phthisis, belongs also in the renal group.

Case 16. (D. S. H. 4168, Path. 226) feared death and *refused food on the ground that she should not eat.* Patient had always been of a despondent and reserved nature (sister also insane) and, after her husband's death, when she was 53, grew unable to carry on her house, dwelt constantly on griefs, entered hospital at 61, and died at 64 ("chronic melancholia"). Death from internal hemorrhagic pachymeningitis. The liver of this case weighed 1074 grams and was fatty. There was chronic interstitial nephritis.

Case 17. (D. S. H. 4707, Path. 498) originally cheerful and frank, lost her situation as companion, grew despondent at failure to get employment, had a "hysterical" attack at 52. It is doubtful whether her beliefs were delusional: "*can never be better,*" "*will not be taken care of,*" "*no place for her.*" "Subacute melancholia." The autopsy showed gastric dilation (over 3000 cc.), *and an atrophic liver and pancreas*, and slightly *contracted kidneys.* The heart was normal. Death from *ileocolitis.* Moderate chronic internal *hydrocephalus.* Dr. W. L. Worcester's microscopic exam-

ination showed rather unusual degrees of *nerve cell pigmentation* (precentral and paracentral).

Case 18. (D. S. H. 8898, Path. 570) was an unmarried daughter of a fire insurance company president. Both her mother and she developed mental disease after the company failed (Boston and Chicago fires). Both mother and father died, and patient was in several hospitals after 36, obscene, denudative, onanist. Delusions concerning *crimes committed.* Satyriasis. *Could hear fire kindled to burn her.* Diagnosis, "*secondary dementia.*"

Death at 54 from *bilateral bronchopneumonia. Atrophic uterus. Cystic right ovary with twisted pedicle: atrophic left ovary: contracted kidneys.* The brain was not abnormal in the gross—but showed (Dr. W. L. Worcester) some acute changes (also larger cells pigmented).

Case 19. (D. S. H. 10106, Path. 663) a cheerful Irish house-wife (mannerism of drawling words) underwent a maniacal attack at 41, and another at 44. Delusions: "*sorry she had lived*": "*broken her religion*" Given to self recrimination.

Autopsy: Death from *hypostatic penumonia. Healed gastric ulcer.* Moderate *arteriosclerosis*, slight *cardial hypertrophy. Granular cystic kidneys. Mucous polyp and subperitoneal fibromyoma of uterus.* The brain was macroscopically normal, but showed superficial gliosis (frontal and precentral) and thinning out of medullated fibers superficially (frontal).

Case 20. (D. S. H. 8963, Path. 679) an epileptic shoe-maker, 50 years, was of the belief that he was *sent to Hospital for hitting a boy* and *was to be executed.*

Autopsy: *Aortic and innominate aneurysm, hypertrophy and dilatation of heart. Interstitial nephritis.* The brain, normal macroscopically, proved microscopically to show, in all areas examined, superficial gliosis. There was gliosis in parts of the cornu ammonis, but no demonstrable nerve cell loss (interesting in relation to the epilepsy).

Case 21. (D. S. H. 4584, Path. 861) cabinet-maker of melancholy temperament, Civil War veteran. Said to have been feeble-minded after six months in rebel prison. Violent at times for twenty years. Did no work, thought "*soul lost.*"

Death from *pneumococcus and streptococcus septicemia.* Chronic diffuse nephritis. The brain was described grossly as normal: but microscopically there was marked superficial gliosis in all areas examined and considerable cell loss in suprastellate layers of precentral cortex. The calcarine sections show little or no cell-loss. But one section from the frontal region is available (right superior frontal). This shows little cell-loss except in the layer of medium-sized pyramids.

Case 22. (D. S. H. 8250, Path. 909) an unmarried woman without occupation, two attacks of "melancholia" at 36, and 40. Always of a retiring and shy disposition. Mental disease began

after father's death. Delusions (if such): *has been selfish and wicked. Constant self condemnation.* Suicidal Exophthalmic goiter.

Autopsy: *Thyroid glandular hyperplasia. Mitral sclerosis. Aortic sclerosis* with *ulceration. Chronic endocarditis. Chronic diffuse nephritis.* Scars of both apices of *lungs*, with small *abscess of left apex.* Emaciation. Brain weight 1050 grams. No gross lesions described; microscopically profound alterations; extreme or maximal cell-losses in small and medium-sized pyramids in both superior frontal regions. Smaller somewhat less marked cell-losses elsewhere.

Upon reviewing the unpleasant-delusion group, then, we exclude one (XII) altogether. It is questionable whether XV actually exhibited delusions at all. We then discover that eight (in all probability all) of our nine remaining cases are renal in the sense of *grossly* evident lesions at autopsy.

But it will be remembered that we transferred three cases originally thought to entertain "not-unpleasant" delusions to the unpleasant group, because their constraint, although conceived to be of divine origin, seemed to be unpleasant (VI, IX, X). Of these VI and X were renal cases; but IX is expressly stated by a reliable observer (the late Dr. W. L. Worcester) to have had normal kidneys as well as heart. In point of fact, however, Case IV had hallucinations and religious delusions ("spirit is here") probably derived therefrom, and Dr. Worcester found an isolated brain lesion correlatable with the hallucinosis; and in any event the emotional state of the patient is in grave doubt.

Accordingly if we take the unpleasant-delusion group to be constituted of Cases VI and X (transfers from the first group), XIII, XIV, and XIV to XXII, that is eleven cases, we come upon the striking fact that virtually all of them are renal cases.

Of course, as (with Canavan) I have been at some expense of time to prove, virtually *all* cases of psychosis (as autopsied) are in a microscopic sense abnormal as to kidneys.[7] But only about a third exhibit *gross* interstitial nephritis, arguing a certain severity of process. The above cases, it will be observed, fall into the *gross* class in respect to renal lesions.

Without laying too much stress on such results, it is worth while to say that, whereas most workers might be willing to surmise that metabolic or catabolic disorder must affect the sense of well-being, I must confess that the discovery of so much gross kidney disease in a group selected on other grounds filled me with a certain surprise.

The literature is not without suggestions as to the possible correlation of renal and mental disorder. Ziehen,[8] for example, remarks that nephritis brings about mental disease in two ways,—through vascular changes which very frequently accompany chronic nephritis and other uremic changes in the blood. Inasmuch as we know that creatin, creatinin and potassium salts irritate the animal cortex, Ziehen notes that psychopathic phenomena may occur in man as a result of slight uremic changes. According to Ziehen, most of these nephritic psychoses run the course of what he calls hallucinatory paranoia (it may be remembered that Ziehen counts among paranoias a number of acute diseases and even so-called Meynert's amentia). Chronic nephritis, as well as acute diabetes and Addison's disease, are thought by Ziehen to produce certain chronic forms of mental defect which he terms autotoxic dementia, but he regards most of these cases as really cases of arteriosclerotic dementia.

It does not appear that Wernicke[9] has considered renal correlations systematically.

Kraepelin[10] mentions the epileptiform convulsions of uremia as well as delirious and comatose conditions, especially those in advanced pregnancy. These uremic conditions may be both acute and chronic. But Kraepelin has not been able to convince himself of the existence of a clearly defined uremic insanity unless the delirious condition just mentioned may be regarded as such.

Binswanger [11]states that the mental disorders occurring in acute and chronic nephritis are either toxemic psychoses on uremic bases, or due to arteriosclerosis. In the latter cases, he states that the disease pictures are as a rule characterized by grave disturbances of emotions, chiefly of a depressive character. He adds that these are all too frequently the forerunners of arteriosclerotic brain degeneration.

A brief mention of renal disease in the general etiology of mental disease is made by Ballet.[12] Ballet states that Griesinger's opinion that renal disease had little importance in the etiology of mental disease and that no one would count the cerebral symptoms of Bright's disease as mental is no longer held. Ballet enumerates a number of works upon so-called *folie brightique* which tend to prove that acute or chronic Bright's disease gives rise either to melancholic disorder or alternately to maniacal and melancholic disorder. How the mental disease is produced is doubtful. Ballet holds that all the various psychopathic disorders resulting from Bright's disease are autotoxic. Renal disease like heart disease is only capable of awakening a latent predisposition or liberating a constitutional psychosis, unless it is merely effecting a species of intoxication.

It cannot be doubted that the relation of kidney disorder to mental disorder is worth intensive study, of which the present communication is merely a fragment. Progress will be of course impeded by the fact that upon microscopic examination, practically all cases of mental disease coming to autopsy show renal disease of one or other degree; in fact, it is perhaps possible to show a higher correlation of renal disease with mental disease than of brain disease to mental disease. Perhaps something can be obtained if we limit ourselves to a study of cases with pronounced somatic renal symptoms and signs, cases with the renal facies and the like.

As to the question of phthisis and mental disease, Ziehen remarks that the tuberculous are often observed to be optimistic but that other cases show a hypochondriacal depression with egocentric narrowing of interests. He speaks of a sort of rudimentary delusional disorder looking in the direction of jealousy in certain cases. Pronounced mental disorder occurs rarely in tuberculosis, according to Ziehen, and leads either to melancholia or to hallucinatory states of excitement, resembling the deliria of exhaustion or inanition. Acute miliary tuberculosis may produce the impression of a general paresis or of an amentia in Meynert's sense. The inanition delirium of tuberculosis resembles that of carcinosis and malaria.

Kraepelin regards tuberculosis as of very slight sig-

nificance in the causation of insanity, despite the fact that slight changes in mood and in voluntary actions frequently accompany the course of the disease. Irritability, depression and sensitiveness, incomprehensible confidence and desire to undertake various tasks, pronounced selfishness, sexual excitement and jealousy are the traits of mental disorder in tuberculosis.

Kraepelin states that many cases of tuberculosis show traits of alcoholic disease and says that the occurrence of polyneuritic forms of alcoholic mental disorder is favored by the association of tuberculosis with alcoholism.

Wernicke does not systematically consider the topic.

Binswanger states that tuberculosis, aside from miliary tuberculosis or meningitis, produces no mental disorder except phenomena of the amentia of exhaustion.

Ballet states that there exists a peculiar mental state in the tuberculous. It is compounded as rule of sadness, of looking on the dark side and of profound egoism. This readily leads to mistrust and suspicion which may be pronounced enough to constitute a sort of persecutory delusional state or a state of melancholic depression (Clouston, Ball). More rarely there are phenomena of excitation explained in part by fever. In its slightest degree this phenomenon of excitation is characterized by a feeling of well-being, of euphoria, which even at the point of death may give the patient the illusion of a return to health, or there may be a more pronounced excitation with impulsive sexual and alcoholic tendencies. Autointoxication may lead to the usual train of confusional symptoms.

If we compare the accounts in the literature of the two conditions here in question, namely, nephritis and phthisis, we must be convinced, that aside from so-called autotoxic phenomena, renal disorder seems to be marked by a tendency to depressive emotions but that phthisis shows not only depressive emotion but also euphoric and hyperkinetic phenomena.

So far as these results thus hastily reviewed are concerned, they are consistent with the appearances in the present group of cases. Both the nephritic and phthisical groups need further intensive study.

As to the question of the spreading inwards or outwards of delusions from the standpoint of the patient, no analysis is here attempted. It is plain, however, that the theopaths, as James calls them, or victims of theomania, to use the French phrase, will be of importance in this analysis because of the equivocal character of the emotions felt in cases of religious delusion.

Summary and Conclusions

The paper deals with delusions of a personal (autopsychic) nature and is one of a series based upon certain statistics of Danvers State Hospital cases (previous work published on somatic, environmental (allopsychic) delusions and those characteristic of General Paresis). The previous work had suggested that somatic delusions are perhaps more of the nature of illusions in the sense that somatic bases for somatic false beliefs are as a rule found. On the other hand, delusions respecting the environment (allopsychic delusions) had appeared to be more related to essential disorder of personality than to actual environmental factors.

The fact that cases of paresis with delusions were found to have their lesions in the frontal lobe, whereas non-delusional cases showed no such marked lesions, is of interest in the light of the present paper because three cases of senile psychosis were found to have delusions of grandeur and, although they are demonstrably not paretic, they also show mild frontal lobe changes supported by microscopic study.

The Danvers autopsied series, containing 1000 unselected cases, was found to show 306 instances with little or no gross brain disease. Of these, 106 had autopsychic delusions and of these 106, 50 cases had delusions of no other sort. 15 of these 50 cases appeared to have been cases of General Paresis in which gross brain lesions were not observed at autopsy, and upon investigation 13 other cases were found to be, for various reasons, improperly classified. The residue of 22 cases was subject to analysis and readily divides itself into two groups of 11 cases each, or two groups of normal-looking brain cases having autopsychic delusions and these only are cases which may be termed the "pleasant" and

"unpleasant" groups, in the sense that the delusions in the first group were either pleasant or not unpleasant, whereas the delusions in the second group were of clearly unpleasant character.

Three of the "pleasant" delusion group were the three cases of grandeur and delusions in the senium above mentioned. Three others were cases of "theomania" in the sense that their delusions concerned messages from God. It is not clear that these three religious cases should be regarded as belonging in the group of "pleasant" delusions on account of the sense of constraint felt by the patients.

The remainder of the "pleasant group," as the delusions were originally defined, turned out for the most part to show either doubtful delusions or delusions involving a sense of constraint rather than of pleasure.

An endeavor was made to learn the relations of pulmonary phthisis to the emotional tone of the delusions. The few available cases in this series seem consistent with the hypothesis of phthisical euphoria (IV, "happiest woman in the world," hearing God's voice, VII and possibly XI).

The problems of the "pleasant" delusion group, as superficially defined, turned out to be a. the problem of a group of senile psychoses with grandiose delusions and frontal lobe atrophy; b. the problem of felt passivity under divine influence; c. the problem of phthisical euphoria.

The group of "unpleasant" delusions in the normal-looking brain group should be diminished by one on account of its positive microscopy (encephalitis). One case (XIII) is a case of mixed emotions of religious type, showing phthisis pulmonalis together with abdominal tuberculosis and nephritis. One case (XV) is doubtful as to delusions; the remainder are subject to renal disease, as a rule associated with cardiac lesions.

Two cases which were transferred from the "pleasant" to the "unpleasant" group on account of constraint feelings, were also renal cases,—VII and IX. The only exception to the universality of renal lesions in this group is the case in which religious delusions were probably based upon hallucinations for which hallucinations an isolated brain

lesion was found, very probably correlatable with the hallucinosis.

Virtually all of the eleven cases determined to belong in the "unpleasant" group are cases with severe renal disease as studied at autopsy.

Whether the unpleasant emotional tone in these cases of delusion formation is in any sense nephrogenic and whether particular types of renal disease have to do with the unpleasant emotion, must remain doubtful. A still more doubtful claim may be made concerning the relation of euphoria to phthisis. The renal correlation is much more striking as well as statistically better based. A further communication will attack the problem from the side of the kidneys in a larger series of cases.

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XLIX

DISSOCIATION OF PARENCHYMATOUS (NEURONIC) AND INTERSTITIAL (NEUROGLIA) CHANGES IN THE BRAINS OF CERTAIN PSYCHOPATHIC SUBJECTS, ESPECIALLY IN DEMENTIA PRECOX*

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I BROUGHT before the Association in 1914 some considerations as to the analysis of cortical stigmata and focal lesions in certain psychoses.[1] I pointed out some promising paths of histopathological research in the cerebral cortex, illustrating my contentions from cases of a disease or diseases clinically known as dementia precox. We are indebted to the cortex topographers—among whom we may name Bolton, Campbell, Brodmann, of the moderns—for the rich lines of research which now open out to the neuropathologist. It has become the duty of the neuropathologist in this new phase of cortex histology to study comparatively the tissues of the "arrival platforms" (sensory), and those of the "departure platforms" (motor), and of the intermediary and more recently evolved tissues of the so-called "elaborative" or "psychic" nature.

Perhaps the best modern description of the new views is to be found in Bolton's recent work on *The Brain in Health and Disease*,[2] with many of whose general contentions most neuropathologists would agree.

* Contribution of the Massachusetts Commission on Mental Diseases, whole number 164 (1916.22). The previous contribution, 163 (1916.21), was by H. M. Adler, entitled "A Psychiatric Contribution to the Study of Delinquency," to appear in American Journal of Criminal Law and Criminology.

My own attention has been attracted by the technical advantages for differential histopathological analysis of cortical tissues of strikingly different architecture lying adjacent to one another. For example, the sensory arrival platform for vision, as represented by the calcarine type of occipital cortex, lies in close proximity to visual elaborative, or so-called "visuopsychic" tissue (to employ Bolton's term), namely, the common occipital type of cortex. These two tissues exist under virtually identical physicochemical conditions. I have found in certain cases a sharp histopathological differentiation in these tissues, which tissues not only exist antemortem under identical conditions, but may be passed through a variety of technical procedures postmortem of identical nature and terminating in material which can be comparatively examined under a single small cover-glass. One of the cases adduced in my former communication proved to show certain chronic changes in the elaborative or visuopsychic tissue, whereas the visual arrival platform or sensory tissue remained without obvious lesion. The case in question clinically showed pronounced and appropriate psychotic symptoms in the shape of certain sharply defined visual hallucinations; whereas the visual function in general was apparently quite normal. Such distinctive correlation between visual hallucinations and visuopsychic tissue lesion is almost (as one might say) too good to be true, and very possibly the hallucinations were really due to some functional involvement of these tissues quite apart from the obvious gliosis of the so-called visuopsychic tissue. The lesions, in fine, may merely be indicators of the true mechanism or chemism of the psychotic symptoms.

Turning attention in 1915 to the technical details of the task of the neuropathologist in the illustrations and interpretation of finer changes, I pointed out the advantages of a truly pathological classification of nerve cells.[3] It appeared that certain apparently homologous and at any rate analogous cell types had surprisingly different survival values. Thus, the large Betz cells, or so-called giant cells of the cerebral cortex, were found exceedingly resistant to certain types of tissue destruction, whereas those very kinds of destructive processes made short work of the large Purkinje cells, or giant cells of the cerebellar cortex. I am bound to say that further

work may show that under some conditions the large cells of the cerebrum and of the cerebellum may live and die, as it were, together, and evince identical survival values under said conditions. With the material at hand, however, I felt sufficiently satisfied to propose a comparative study of the differential viability of the various nerve cell types.

I seemed to see as a construction of the future an essentially "pathological" classification of nerve cells on the basis of their powers of resistance or survival values. I thought that the neuropathologist might well proceed to collect data as to the differential effects of simplification or decomplication of nerve tissues. Are there not, however, cases always outstanding in which there are no such changes; psychoses in which, namely, the brain and especially the cerebral cortex is acting abnormally but shows no structural sign of its abnormal action? Are there not, in short, cases in which the brain is theoretically and intrinsically entirely normal and merely acting abnormally under the stress of conditions exogenous to the nervous system? This is the problem of normal-looking brains in the psychoses, contributions to which have been made by several workers in Massachusetts institutions for some three or more years past.[4-8]

Are there cases in which, however carefully we shall study the elaborative tissues alongside the arrival and departure platforms, we shall not find lesions of importance? Are there cases in which the problem of the survival values of different types of nerve cells will not be at all in play? Is there anything in the neuropathological literature or elsewhere which goes definitely to prove that the clinical phase of a psychosis is identical with a phase of cell injury? Is it certain that the period when a patient shall seem mentally altered to a skilled observer is identical with a period in which coarse or fine anatomical methods shall demonstrate important structural lesions? Is there not, in short, a group of cases in which the solution of the psychotic problem is entirely removed from the zone of cortex histology, and even theoretically of cortex chemistry and physics, simply because the cortex is presumed to be working under quasi normal conditions? Is there not a problem of the psychoses which we may term the problem of "discords

played on good instruments?" Unless some answer to this question can be given, the theory of the genesis of the psychoses is likely to be at loose ends.

Accordingly I felt it of strategic value in this work to study a group of normal-looking brains, to isolate if I could a number of convincing examples of *psychoses of definite and relatively permanent nature, in which cases pertinent lesions should be absent or negligible.* I had forthwith to disregard great numbers of brains with a number of lesions, not because I was entirely sure that these lesions were correlated with the psychoses, but because they clouded the issue of determining absolute normality for the brains. Even on the standards of the autopsy table, something like two-thirds of the brains that accrue from a hospital of committed types of mental disease exhibit more or less pronounced gross lesions, largely of an atrophic nature or of such a nature that the differential diagnosis between acquired atrophy and inborn hypoplasia cannot be made. If the neuropathologists at work are a bit structuralistic, then, as in our Danvers material, about three-fourths of the brains turn out to be abnormal in some important way. The finer structural diagnosis yields in this event still more cases of an atrophic or hypoplastic nature, and tends to exhibit a remarkable preponderance of such conditions in the frontal lobes. In fact, the frontal lobes in the hands of some observers are found atrophic or hypoplastic in greater or less degree in two-thirds of relatively long series of brains.

In several years' work I developed certain criteria of the normality of the brain which may be in part within the personal equation, but which, applied to a Boston State Hospital series (in the hands of my colleague, Dr. M. M. Canavan), left only one-eighth of all the brains of a series of 145 subjects without gross lesions of apparent significance, or of such a nature that it would be dangerous to say that the lesions were without relation to the psychoses. Whether cystic, atrophic, hypoplastic, or otherwise characterized, the lesions in seven-eighths of the Boston series were too perturbing to deal with in the problem of "discords played on a good instrument."

Nineteen of the 145 brains studied at the Boston State Hospital in the years 1910 to 1913 by Dr. M. M. Canavan and the writer

turned out to be of a normal appearance in the gross (11.7 per cent.). These 19 cases were especially studied as to their clinical histories. Their previous histories and hereditary features were particularly looked into by a trained worker in eugenics (namely, Miss Anna E. Steffen) who went into the field after new data to correlate with the cases viewed in their entirety. Moreover, all the brains systematically photographed by Mr. Herbert W. Taylor under the supervision of Dr. Annie E. Taft, both before and after the removal of the pia mater, so that the upper, lower, lateral, and mesial aspects of each brain could be readily compared side by side.

Orienting microscopic examination was then made of a few areas in each of the 19 cases. One of the cases, for example, turned out to be microscopically a case of general paresis; another was very probably a case of the so-called central neuritis, with a variety of additional microscopic changes. Still another case showed striking universalized cell changes, also of an acute nature. Other cases showed convincing degrees of diffuse cell loss or of diffuse arteriosclerotic change of such a nature that one could hardly understand the alleged normality of the brains as observed at the autopsy table. A few of the cases were of brief duration and clinically of such a nature that it might be doubted whether they would upon survival have turned into cases of chronic mental disease. The clinical histories of some cases indicated such brief illness that it might well be doubted whether the brains would register gross atrophic changes in the time available.

However, in the course of the successive application of higher and higher powers (as it were) of logical analysis, we arrived at a set of 5 cases, which cases were all clinically of a sufficient duration to warrant the idea of relatively permanent mental change, had all been observed with adequate accuracy, could be fitted sufficiently well into prevalent classifications of mental disease, and *still failed to show, either at the autopsy or in the systematic photographic analysis of the cortical appearances, or even in the orienting microscopic examination of a number of areas, any convincing evidence of brain disease.* It is not that the microscopic examination failed to show changes, and possibly changes of some importance (as will be mentioned below); but the microscopic changes found in the orienting analysis

of these 5 brains were, after all, hardly striking enough to warrant correlation with the symptoms observed or the entities determined.

It seemed that these 5 cases, the product of successively more intensive analysis of a large series, might permit us to draw the lines a bit closer about the classical position of the psychiatrists as to generally functional psychoses. In any event, the classical position of various psychiatrists as to the functional psychoses is often based upon the vaguest of formulæ, perhaps even in some cases on the private ontological wish of some one that mechanistic or vitalistic views of mental disease shall prevail. It is clearly our duty in this situation to discover what the facts are about the very existence of these genuinely functional psychoses, the psychoses that proceed with utter intrinsic normality of the brains which are running along just as a physiologist might wish them to run.

I am far from holding that the observation of numerous minor lesions in a variety of loci in all of the brains in question proves that no brain in a psychopathic subject is structurally normal. No one could be so naïve as to suppose that association or correlation of lesions and symptoms amounts to a causal nexus between the lesions and symptoms. Still, I am bound to say that the functionalist wishing to prove that mental disease is consistent with a structurally normal cortex must find himself greatly at a loss when confronting the present material. Even with all due allowance for the personal equation in histological analysis (and only histologists are aware how wide is this margin), the changes found, varying as they do from brain to brain and from locus to locus within a given brain, must certainly be given due consideration. I conceive that no brains of normal non-psychotic subjects have so far been examined with equal thoroughness.

Without insisting upon gaining a decision in the struggle over intrinsically normal brains in the psychoses, and conceding the necessity of more material than here presented, I wish to offer some interesting considerations that came rather as a by-product of this work. These considerations I had intended to indicate in the title of this communication. The dissociation of parenchymatous and interstitial lesions, set forth in my title, will remind the pathologist of the older contentions concerning diseases, *e. g.*, of

the kidney. Such, for example, is Senator's classification of chronic forms of kidney disease. In point of fact, the cases of brain disease here studied do rather clearly show a tendency to pure parenchymatous disease on the one hand, to pure interstitial disease on the other hand, and to a mixture, or "diffuse" condition in one case. Of course, when it comes to kidney disease, the pathologist has often felt it a merely pious wish that chronic Bright's disease shall fall at all easily into parenchymatous and interstitial forms. Nor do I feel that the cerebral cortex or congeries of organs, still more labile than the kidney, is likely to permit a similar classification to click easily into place.

We must clearly consider that the scar stage of cortex diseases will complicate any picture. Equally when Volhard and Fahr, in their well-known monograph on Bright's disease, attempt to separate the nephroses from the nephritides, they are compelled to find scar stages in both types.[9]

The peculiar advantage for our present purpose of these 5 cases is that our study has proved them to be not yet in a scar stage, if indeed they were ever destined to become visibly and tangibly atrophic. Here are cases which have proceeded for a number of years without evidence of naked-eye change and without palpable changes to the finger. Microscopically, however, they prove to be in some instances the polar extremes of one another; a case with highly marked degenerative cell changes proves to show entirely negligible neurogliar changes. Another case with extremely marked neurogliar changes (though without induration in the gross) proves to have little or no parenchymatous change in the shape of cell degeneration.

Let us look more in detail at these changes. The following is a list of cases having brains normal looking in the gross, both as observed upon the autopsy table and after photographic analysis, and yet microscopically normal or with negligible or minor changes in eight orienting areas of the cerebral cortex.

Case.	Sex.	Age.	Onset.	Duration.	Diagnosis.
11.36 . . .	F.	31	29	20 months	Manic-depressive.
12.47 . . .	F.	27	25	2 years	Dementia precox.
13.7 . . .	F.	64	20	3½ years	Manic-depressive.
12.41 . . .	F.	60	50	10 years	Paranoia.
10.9 . . .	F.	56	42	14 years	Dementia precox.

The microscopic examinations were, although extensive, limited to observations of material prepared in two classical ways, namely, by a monochromatic nuclear stain demonstrating cell nuclei and cell bodies with sufficient clearness, and by the Weigert myelin sheath method. Notes were made of appearances in the following areas on each side: prefrontal, superior frontal, precentral, postcentral (taken superiorly), postcentral (taken inferiorly), superior parietal, angular, Broca, transverse temporal, superior temporal, middle temporal, insular, gyrus rectus, pyriformis, calcarine, making 16 areas on each side, or 32 areas in all. Owing to technical difficulties, occasional areas could not be completely studied and were omitted.

Notes were made as to the appearances, as a rule, in seven zones of the cortex and in the underlying white matter. The resulting tabulations included 144 to 232 histological observations upon nerve cells, neuroglia cells, satellite cells, axonal reactions, chromatolysis, pigmentation, arterial changes, etc., in separate loci defined by area and layer. An endeavor was made to include among nerve cell changes, besides axonal reaction and pigmentation, also the somewhat more difficult diagnostic points of cell swelling, cell shrinkage, diffuse staining, deep staining, pale staining, and the like, some of which changes are, of course, difficult of interpretation, and are even within the range of the personal equation.

The following table shows the proportion of lesions noted in the loci observed in these 5 cases.

Case.	Diagnosis.	Duration.	Loci examined.	Lesions noted.	Ratio of lesions to foci.
11.36	Manic-depressive	20 months	152	111	73
12.47	Dementia precox	2 years	232	193	83
13.7	Manic-depressive	3½ years	144	121	84
12.41	Paranoia	10 years	208	149	71
10.9	Dementia precox	14 years	224	122	54

It is clear that these so-called *lesions* are of various nature and of very varying significance to the psychoses in question. Accordingly a collation of the lesions noted has been made to show the proportion of what we regard as nerve-cell losses and of what we regard as proliferative changes. We freely grant the difficulty of a diagnosis

of nerve-cell losses upon qualitative grounds and without adequate measurement and possible photography. However, we present our analysis for what it is worth:

Case.	Diagnosis.	Duration.	Loci observed.	Nerve-cell losses diagnosticated.	Ratio of supposed nerve-cell losses.	Evidences of neurogliar proliferation observed.	Ratio of proliferative changes.
11.36	Manic-depressive	20 months	152	66	43	12	7
12.47	Dementia precox	2 years	232	123	53	7	3
13.7	Manic-depressive	3½ years	144	33	22	55	38
12.41	Paranoia	10 years	208	91	43	24	11
10.9	Dementia precox	14 years	224	25	11	19	8

With all due concession of the difficulty of making a qualitative estimate of nerve-cell loss, it is entirely clear that these cases tremendously differ among themselves in the matter of nerve-cell loss as we render the diagnosis. Compare for example the 11 per cent. of nerve-cell losses in Case 10.9 with the 53 per cent. of loss noted in the various loci of Case 12.47. Almost any histopathologist would readily agree that it is far easier to make a diagnosis of neurogliar proliferative changes than of nerve-cell losses on account of the fact that in cellular gliosis we deal with positive added factors in the shape of clearly observable nucleated cells. It is clear from the great disparity in percentages of proliferation as compared to cell losses either that gliosis does not run *pari passu* with nerve-cell losses or that our diagnosis of nerve-cell losses errs in the direction of finding too many losses. However, just as in the case of the nerve-cell losses, so also in the case of the neurogliar proliferative changes, we find a great range of differences; thus in Case 12.47, we find but 3 per cent. of proliferative changes whereas in Case 13.7 we find 38 per cent. Without closer analysis, it is clear that Case 12.47, having the highest percentage of nerve-cell losses (53), is precisely the case which has the lowest percentage of proliferative neurogliar changes (3). It is clear that Case 11.36 and Case 12.41 tend in the same direction since Case 11.36 shows 43 per cent.

cell losses to 7 per cent. neurogliar proliferative changes and 12.41 shows 43 per cent. of nerve-cell losses but 11 per cent. of neurogliar changes. On the other hand, Case 13.7 shows the opposite tendency, for it contains the highest percentage of neurogliar proliferative changes, namely, 38 and next to the lowest percentage of nerve-cell losses, namely, 22. Conditions in these two directions of nerve-cell loss and neurogliar proliferation are numerically about even in the fifth case, namely, 10.9.

A number of tentative conclusions might be drawn from these cases but it is worth while to analyze the findings more in detail and to examine the clinical histories before rendering these conclusions. First, let us present in tabular form some details of the microscopic findings in these cases. The columns deal with the white matter, the undermost or fusiform layer of the cortex and then in succession the layers of internal large pyramids, stellate cells, external large pyramids, medium pyramids, small pyramids and the plexiform layer. The findings in regions like the occipital which do not entirely agree in general construction and nomenclature with the other areas of the cortex have had their findings listed under the headings of those areas which are most nearly homologous with the areas just listed.

The abbreviations in the columns are as follows:

axre	= axonal reaction.
artscl	= arteriosclerosis.
cegl	= cellular gliosis.
chrly	= chromatolysis.
irreg	= irregular grouping of cells.
l	= loss.
perigli	= perivascular gliosis.
perivdep	= perivascular cell deposit.
pig	= pigmentation.
sacpig	= saccular pigmentation.
satc	= satellite cells.
shr	= cell shrinkage.
sm	= small cells.
vth	= vessels thick.

I hope to publish elsewhere the details of the clinical, anatomical, and histological examinations of these 5 cases, but for the present purpose I will mention only a few of those features which seem particularly pertinent to the question of the dissociation of parenchymatous and interstitial lesions.

	White matter.	Fusiform.	Internal large pyramid.	Stellate.	External large pyramid.	Medium pyramid.	Small pyramid.	Plexiform.
11.36 Manic-depressive 20 months	3 perivdep 1 perivgli 5 vth	1 axre 2 satc 10 l 1 irreg	3 axre 1 chrly 1 satc 1 shr 12 l	1 satc 1 shr 10 l 3 irreg ..	4 axre 2 satc 1 shr 10 l ..	1 axre 2 cegl 1 satc 1 pig 1 shr 13 l	2 cegl 11 l 3 shr 1 vth 1 irreg	0
	9	14	18	15	17	20	18	0 = 111
12.47 Dementia precox 2 years	12 artscl 1 l 1 vth	13 axre 1 satc 15 l 1 sacpig	3 axre 2 chrly 1 sacpig 1 irreg 3 shr 2 satc 17 l	4 axre 1 shr 23 l	5 axre 1 chrly 4 shr 18 l 1 sm	1 axre 1 irreg 4 shr 24 l 1 sm	1 cegl 25 l 2 shr 1 sm	3 cegl
	14	30	29	28	29	31	29	3 = 193
13.7 Manic-depressive 3½ years	3 pig 1 perigli 1 cegl 6 vth	5 satc 3 shr 2 pig 7 l 3 cegl	4 satc 2 axre 6 pig 5 l 1 shr	2 satc 1 cegl	1 axre 8 satc 5 pig 2 cegl ..	11 l 3 satc 5 cegl 2 shr 1 irreg	10 c 4 cegl	17 cegl
	11	20	18	3	16	22	14	17 = 121
12.41 Paranoia 10 years	2 perivdep 8 artscl 1 pig 2 l	1 axre 1 cegl 8 satc 3 shr 2 sm 16 l	6 axre 3 shr 1 sm 14 l ..	3 axre 3 satc 15 l	1 sm 2 shr 14 l 2 axre 3 satc	4 axre 1 satc 1 shr 15 l	4 cegl 3 shr 15 l 1 satc	3 cegl 1 vth
	13	31	24	21	22	21	23	4 = 159
10.9 Dementia precox 2 years	6 pig 9 vth 3 perivgli	5 axre 9 satc 2 shr 2 irreg 3 pig 2 l	4 axre 3 pig 10 l 1 irreg 3 shr	4 axre 2 satc 1 vth 3 irreg 3 l	7 shr 3 satc 4 irreg 3 l 2 pig	4 axre 1 pig 4 satc 2 irreg 3 l	5 axre 1 irreg 1 shr 4 l 1 vth	1 vth 1 pig
	18	23	21	13	19	14	12	2 = 122

Let us consider, first, Case 12.47, that case in which the parenchymatous change was maximal and gliosis minimal. The cell lesions in this case were observed in 53 per cent. of all loci examined. Gliosis appeared in but 3 per cent. of all loci examined; that is, 123 examples of cell loss were noted in 232 loci; whereas but 7 instances of neurogliar proliferation were observed in these same loci. This case was one of dementia precox and was the youngest of these cases: an Irish servant girl, with some drunkenness and tuberculosis in the family, herself a victim of tuberculosis, and in point of fact developing symptoms after discharge from a sanatorium for tuberculosis; a case with two years of mental symptoms, dying

at the age of twenty-seven years. The autopsy showed sclerosis of the aorta and of the splenic artery; the heart muscle showed brown pigmentation, and the vascular vessels were small. Even the brain was not destitute of minor arteriosclerotic changes, more marked than in any other case of this particular series of 5. A bird's-eye view of the lesions would, in point of fact, suggest a greater age than the patient actually had reached. At first, apprehensive, depressed, slightly slow in reactions, the patient after some weeks became apathetic and unwilling to take food, became mute or whimpering, and finally completely non-coöperative, sitting for long periods in a particular attitude, and showing a characteristic and marked flexibilitas cerea. From this condition she fell into a sort of catatonic stupor, from which she emerged some weeks later in a euphoric state, able to feed herself and with a good appetite, but with loss of memory for recent and remote events and disorientation. Some months later, after headaches and dizziness and a fainting spell, the patient took to bed in what seemed to be a catatonic stupor, from which, however, she once rose and moved about.

If there is such a disease as dementia precox, it is probable that this case must be counted therein. Two years is probably a safe period in which to allow the development of atrophy, or at all events of a certain induration of the brain, provided that there were any tendency on the part of the tissues to gliosis. In but 7 loci out of the 232 examined was there any evidence of gliosis, and these evidences were for the most part examples of cellular gliosis in the outmost layers—a locus of election for such changes in a variety of diseases. Are we not here dealing with a comparatively pure example of mild but exceedingly wide-spread parenchymatous changes without tendency to neurogliar reaction? It is, of course, hard to believe that this case, had it lasted twenty instead of two years after onset, would fail to have shown far more gliosis. Just as the so-called nephrosis of Volhard and Fahr may pass into a scar stage of a secondary nature, so we may suppose that the brain tissues of Case 12.47—a case with maximal evidence of cell loss—might well pass into a stage of corresponding interstitial loss. I pass over comments concerning the correlation of particular symptoms with the distribution of lesions in particular loci, as without the scope of this paper.

Let us now throw into contrast with Case 12.47 that case (13.7) which showed the maximum degree of neurogliar change (38 per cent. of loci examined) and far less evidence of cell loss (22 per cent. of loci examined). This is a case, aged sixty-four years at death, diagnosticated manic depressive psychosis of a total duration of three and a half years, occurring in two attacks: one, an attack of so-called slight melancholia, a year in duration, beginning at twenty years of age; and the other before her death. The family, in this case, showed a number of instances of cardiovascular disease, and the abdominal aorta of the patient herself was found altered into a calcareous tube, with ulcers, vegetations, and a thrombus. In the head, the patient showed basilar arteriosclerosis; but the brain tissues so far as examined showed little or no evidence of fine arterial changes. In fact, the high percentage of gliosis cannot be related to vascular change with any probability. The gliosis is pretty generally distributed throughout the loci; many instances of cellular gliosis are found in the outermost layers, but satellitosis is especially marked in the inner layers, associated with more or less cellular gliosis. It is easy to claim that the gliosis is a phenomenon of age, and such it may actually be. The ordinary hypothesis for its production would be the loss of cells with the endeavor on the part of the neurogliar tissue to replace these cells. Bearing in mind that the brain was of normal appearance and weight (was, in fact, something like 200 grams overweight in relation to the body length: 1370 grams), we are confronted by the fact that the evidence of cell loss in the cortex is slight. If we are to suppose that the long lucid interval of decades between the attack in the twenties and the patient's death in the sixties ran on without special evidence of cell loss, and regard the mental disease as in nowise due to structural cell changes, then, perhaps, we may think of a progressive, slight but wide-spread, gliosis going on without special relation to the symptoms of the case. If we omit reference to the gliosis altogether, and compare this case (13.7) with the above-mentioned case (12.47), we perceive that a patient, aged sixty-four years, with three years and a half of symptoms, two years and a half immediately preceding death, exhibits but 22 per cent. of cell losses in loci examination as compared with 53 per cent. in a patient dying at twenty-seven

years after two years of symptoms. On the basis of parenchymatous change alone, accordingly, this oldish case of manic-depressive psychosis (13.7) is better off in nerve-cell content than a far younger case of dementia precox. Yet in both instances the play of lesions in the parenchyma has gone on without a proportionate amount of neurogliar change. The severely affected parenchymatous case (12.47) has little or no gliosis, and the far less severely affected parenchyma of Case 13.7 is supplied with a gliosis which we may possibly attribute in part to the mysterious operations of senescence.

A third case (11.36) is another case of comparatively brief duration, some twenty months or two years, with death at thirty-one. This case should be worth comparing with 12.47, having two years of symptoms with death at twenty-seven. This case shows somewhat less parenchymatous change: 43 per cent. in the loci examined, and somewhat more neurogliar change: 7 per cent. of loci examined. The first fact which attracts us in this case is the diagnosis: manic-depressive psychosis. This diagnosis was grounded upon the obvious mania of the patient on admission. Nevertheless, the patient herself described auditory hallucinations—a symptom of somewhat ominous character, and not often found in clearly defined form in good examples of manic-depressive psychosis. Again, the patient executed a variety of impulsive acts and showed stolidity and indifference in certain phases, together with persistent slight silliness, which may well be regarded as consistent with the diagnosis of dementia precox. The fact that the patient also showed symptoms characteristic of manic-depressive psychosis is not at all inconsistent with the theory of these diseases, since virtually every symptom of manic-depressive psychosis is nothing but an exaggerated or lessened degree of a normal phenomenon. Hence, as we currently say, dementia precox patients as well as all other mental patients may show manic-depressive symptoms; whereas the true manic-depressive patient fails to show anything but normal phenomena in abnormal degree. It would be hardly convincing to alter the diagnosis of manic-depressive psychosis to dementia precox in the light of cortex histology in the present dubious phase of this topic. I am inclined to feel, however, on the clinical data alone, that the case is one of dementia precox and that we would best

align Case 11.36 with Case 12.47 as cases developing a variety of dementia precox symptoms in the twenties and both succumbing to tuberculosis. Both showed cardiovascular tendencies; both were servant maids; both had a certain family taint: drunkenness and tuberculosis in one; neurosis, heart disease, diabetes, and tuberculosis in the other. Of course we may give ourselves leave to inquire whether the tuberculosis in these cases has a genetic relation to the mental disease or possibly to the parenchymatous changes in the brain. At all events there is something peculiar and interesting in these cell losses which are not accompanied by gliosis.

Another case (12.41) has as high a proportion (43 per cent.) of parenchymatous changes as did Case 11.36, although it possesses a slightly higher ratio of neurogliar changes (11 per cent.). Here is a case with symptoms of ten years' duration, with death at the age of sixty years. If it was easy to ascribe to old age the 38 per cent. neurogliar changes in Case 13.7, a woman dying at sixty-four years, it is remarkable that senescence has produced few neurogliar changes in Case 12.41. Moreover, in Case 12.41, a great many of the neurogliar changes are of the nature of satellitosis, changes, namely, which are rather more related with cell loss than with any senescent tendency to a general induration of the tissues. The diagnosis in this case is not too easy, although a delusional condition was paramount and the diagnosis paranoia or paraphrenia (in the newer Kraepelinian sense of this term) may be regarded as safe. The patient's father was dissolute and she early had illegitimate children, but thereafter her progress was upward, and upon refusal of her paramour to marry her, she left him and became active in church work, and in the course of years began to present an appearance of refinement. In the fifties, however, she became mildly deluded about her pastor, circulating stories of misconduct and talking of his influence upon her. After threats on his life, she was committed. She held the belief that the pastor had hypnotized her and had caused many deaths in his church.

This case, like Case 12.47 and 11.36, was also extensively tuberculous, having developed tuberculosis late in life.

Here, then, are three cases: Case 12.47, Case 11.36, and Case 12.41, aged at death twenty-seven, thirty-one, and sixty years

respectively, with tendency to marked parenchymatous change associated with but slight neurogliar changes, all three belonging very possibly to the group of endogenous deteriorations in the sense of Kraepelin; the first undoubtedly a victim of dementia precox, the second very probably so, and the third an early sex delinquent, later hyperreligious, becoming paraphrenic or paranoiac. When contrasting these three cases with a case pretty certainly not one of the so-called endogenous deteriorations, namely, Case 13.7, a case which showed two phases of manic-depressive psychosis, we have thrown into strong contrast the parenchymatous changes of our three "endogenous" cases and the lesser development of cell losses in the manic-depressive psychosis.

There remains for consideration one case—the most nearly normal case of our whole series—a case dying at fifty-six years of age, after fourteen years of frank symptoms, which received the diagnosis of dementia precox. This patient was of noble French extraction, and had a high-school education. She gradually developed from an apparently genuine jealousy a wealth of paranoid ideas about her family and acquaintances. She was given to extravagant accounts of her delusions, and went at times out of the hospital ward dressed in brilliant colors, youthfully, and with ribbons in her hair, and wore a lace coat of peculiar pattern. She was for years a striking patient, decorating herself with buttons, arranging her hair in small curls, and talking continuously and fantastically. She later developed auditory hallucinations and a number of hypochondriacal ideas. The cell losses in this case came to but 11 per cent., and the gliosis to but 8 per cent. Accordingly, if our method of judging lesions is correct, this case is the most nearly normal case in the series. There are some interesting points which may be published later concerning the distribution of lesions in different loci in these cases. From the present point of view, I wish to call attention merely to the distinction of this case from the other cases which we should perhaps term also cases of endogenous deterioration. I may call attention to the absence of active tuberculosis from this case.

Thus, in our whole series, the youngest patient (Case 12.47) showed the most instances of cell loss and the least instances of

neurogliar proliferation. On the other hand, the greatest number of instances of neurogliar proliferation occurred in the oldest patient (Case 13.7). Age alone, however, can scarcely account for the gliosis in Case 13.7, since another patient almost as old (Case 12.41) showed very little gliosis. The neurogliar changes are very possibly dissociated from the parenchymal changes.

If we turn from neurogliar changes to nerve-cell changes, we find that a case of manic-depressive psychosis (Case 13.7), although sixty-four years of age and the victim of a variety of somatic diseases, showed far less cell loss than 3 cases (Cases 12.47, 11.36, and 12.41) apparently belonging to the group of so-called endogenous deteriorations. We might at first sight suppose that the cases of endogenous deterioration are more prone to parenchymatous changes than to neurogliar changes. This may well be the case, but we must remember that these 3 cases also happen to be victims of severe tuberculosis. Moreover, we have another case (Case 10.9) and that the case of longest duration in the whole series (fourteen years), which not only showed a low percentage of neurogliar changes but a low, and in fact, the lowest percentage of parenchymatous changes. This most nearly normal case in our series was not actively tuberculous. It was most probably a case of endogenous deterioration. The changes in this case were singularly localized in certain regions, so that if the process exhibited in some loci were spread over the entire nervous system, the case would undoubtedly resemble the others of the endogenous series.

I cannot enter here the unsolved question of the relation of tuberculosis to dementia precox and the possibility of lytic changes somehow incidental to tuberculosis taking place in the nervous system. I am limiting the argument to the question of the dissociation of parenchymatous and interstitial changes.

We have thus considered:

1. The case with most instances of cell loss (Case 12.47).
2. The case with least instances of cell loss (Case 10.9).
3. The case with the most instances of gliosis (Case 13.7).
4. The case with the least instances of gliosis (Case 12.47).

And we have learned that there may be a marked lack of correlation between cell loss and glia proliferation (see especially Case 12.47).

The two remaining cases (Cases 11.36 and 12.41) resemble each other in both proportions (43 per cent. to 7 per cent., and 43 per cent. to 11 per cent.).

Three of the cases with large degrees of cell loss had no corresponding gliosis. These were cases probably belonging in the dementia precox group. They were all three very actively tuberculous.

The most nearly normal case of all was one that may perhaps be called paranoia. There was no active tuberculosis in this case. The lesions were exceedingly focal, though of the same general appearance (cell losses in a variety of loci) as those of the more wide-spread parenchymatous lesions.

To sum up:

1. Parenchymatous (neuronic) lesions and interstitial (neuroglia) lesions may be dissociated and combined, much as similar lesions in the kidney.

2. A case of manic-depressive psychosis failed to show convincing degrees of parenchymatous lesions.

3. Dementia precox cases had marked parenchymatous disorder, to which gliosis was not at all proportionate.

4. It is necessary to find and study by like methods a good group of non-tuberculous cases of dementia precox, so as to exclude tuberculosis from having a share in the production of these lesions.

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FOCAL LESIONS OF THE CORTEX OF THE LEFT ANGULAR GYRUS IN TWO CASES OF LATE CATATONIA.*

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ABSTRACT.

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I. Introduction.

The reasons for reporting these cases are several and distinct. Of course, we mean by our title to suggest a genetic relationship between the focal lesions and the subjects' symptoms. Yet we cannot offer proof of such relationship and are in one sense merely hoping to excite others to opposition or to similar reports. In the next place, however, it is undeniable that every case of truly focal lesion in any portion of the brain's silent areas needs reporting for the purposes of future compilation. And, in particular, lesions of the angular gyrus merit attention because of the very various claims made by experimenters and clinicians concerning its function.[1] Lastly, the fortunate peculiarity of the present lesions—their superficiality and virtual limitation to the grey matter—suggests a special value for these cases in proving the almost purely associational (non-projective) nature of the angular gyrus.

First, concerning the possible relationship between the lesions and the symptoms, we may call attention to the fact that the lesions lodge well within the confines of the posterior association area of Flechsig and that the lesions appear to be accompanied by no structural disorder of the projection system. Hence we might well be entitled to consider that the functional results of these lesions would either be *nil* (or negligible as amounting merely to an undemonstrable minor memorial or conceptual defect) or else in some sense mental. If mental, we might naturally suppose some disorder of memory or of conceptual power, possibly some functional loss which would contribute to a form of aphasia or other disorder of the thought-speech mechanism (see below).

Perhaps what most attracted us to the analysis of these cases was the possibility that they would contribute to the statistical conclusions put forward by Southard in 1910[2] and in a measure confirmed in 1915[3] concerning the relationship between catatonic and parietal lobe lesions. We may recall that Southard found that some *86 per cent* of his series of *dementia præcox cases showed lesions* and that four groups of cases could be separated out on the basis of focal atrophies and sclerosis in particular areas. One of these groups was a group termed post-Rolandic and included cases with lesions in the postcentral and superior parietal regions and in the occipital region. It was those cases with *postcentral and superior parietal lesions* which showed catatonia. Likewise,

at that time Southard described a cerebellar group with catatonia (recalling the not exactly similar theoretical contentions of Kleist[4]) and a small group of infra-Sylvian cases (too small for correlations, although this gap has since been made good by a more extensive analysis of new cases, 1915). Then there was a good-sized group of pre-Rolandic or frontal cases with paranoic features predominant.

That 1910 series was, on the whole, rather deficient in cases with lesions in the more inferior portions of the posterior association area, and we were by consequence watching for such.

No one was more surprised than the physician most acquainted with the first case clinically, Dr. S. W. Crittenden, to learn at autopsy that the case was one of cerebral cyst of softening, since (barring the initial fainting spell) there had been no features to suggest cerebral arteriosclerosis, and the diagnosis had lain between *involution-melancholia* and *catatonic dementia præcox*. In fact, had it not been for the well-established onset at 43 years of age, there might not have been the slightest doubt of the propriety of the diagnosis *catatonia*.

Curiously enough, when the second case came to autopsy, the physician in charge, Dr. Wm. W. Dobson, remarked that this case was one of dementia præcox and in view of the contentions of one of the writers as to the parietal correlations of dementia præcox, as well as the results in the first case, inquired whether we might not also in this case find an angular gyrus lesion. The astonishment of all may be imagined when the tubercle was found in the appropriate place.

We may recall that the 1910 series of dementia præcox cases was so drawn as intentionally to exclude all cases of a decidedly "organic" appearance. Thus, had the present case been autopsied at Danvers during the years just preceding 1910, it would *not* have been used to build up the percentage of 86 having focal atrophies or scleroses. In fact eight cases of dementia præcox were actually excluded from the analyzed Danvers material on the score of marked arteriosclerotic changes (as well as five with marked generalized brain atrophy and 11 with marked chronic diffuse leptomeningitis). Some undoubted and beautiful cases of dementia præcox of a group to which the present case may belong were thus excluded from the 1910 analysis in which *complications* had

rigorously to be shunned. Some day that more "organic" series of dementia præcox cases should be carefully analyzed. Meantime the present case may serve as an example.

What is *late* catatonia? No extended analysis of the literature is necessary for the present note, and we will content ourselves with abstracting Kraepelin's most recent statements. Kraepelin states (1913) that 3.3 per cent of his series of over a thousand (1054) cases of dementia præcox took their rise between the 40th and the 45th year (also 1.2 per cent between 45 and 50, 1.1 per cent between 50 and 55, and 0.2 between 55 and 60).[5] Kraepelin mentions Petrén's 24 cases of catatonia with onset after 40 (including six between 50 and 55, as well as one at 58 and one at 59). Schröder also reported a case with onset at 59. Zweig reported five cases with onset after 40. Schröder reported 16 cases of *Spätkatatonic* (earlier attacks in four).

Those involutional cases which Kraepelin regards (1913) as most open to the suspicion of being dementia præcox are characterized by an onset with *apprehensive excitement* and *depressive delusions* together with *catatonic signs, automatism, inaccessibility, resistiveness, stereotypy of posture and movement.* These cases then speedily terminate in pronounced mental deterioration (occasionally there may be a transient period of improvement at first). Kraepelin also mentions certain paranoid forms which may seem to warrant the diagnosis late catatonia. Kraepelin has no decisive word on this topic and relegates most of the pertinent discussion to his chapters on presenile and paranoid mental disease.

Turning to Kraepelin's latest work on presenile mental disease (1910) we find late catatonia considered as very probably a disease of quite different stamp from the common earlier type of catatonia.[6] "So long as we are completely in the dark as to the causes and nature of catatonia," says Kraepelin, "it cannot be denied that the process which comes on as a rule in youth may sometimes set in later—a point for anatomy to decide." Whereas formerly Kraepelin held that the climacteric might produce the same sort of results as adolescence, he has latterly become convinced that the late cases do not show the same sort of structural changes as the early cases.

To the above sketched clinical description, Kraepelin adds a few other clinical features in these late cases, *e. g., rhythmic movements, impulsive acts, incoherent, disconnected talk,* and says that,

in addition to excited and apprehensive delusional states, there may be cases showing gayety and delusions of grandeur.

It will be shown below that our cases fit fairly well with the main lines thus drawn by Kraepelin for late catatonia. However, inasmuch as at autopsy in the first case, we arrive (however unexpectedly) upon cortical arteriosclerosis, it may be well to summarize from the same Kraepelinian source (1910) the clinical findings in arteriosclerotic insanity.[6] It will require no thorough study of our case to show how far removed it is from the usual frame of arteriosclerotic insanity.

There are, according to Kraepelin, two main groups of arteriosclerotic mental diseases: 1. A group in which the total psychic *personality* becomes gradually altered, long before there is evidence of mental defect (some of these cases are epileptoid; some progress slightly, others more rapidly; all are rather states of mental weakening than severe dementias); 2. a group in which *sharp seizures* set in early and *paralyses* occur but *mental changes* remain slight (these cases suggest lesions in the larger vessels) until years have passed.

Our first case must surely fall into the former group clinically, if into either group; yet the brain lesion would rather suggest the second group. As a matter of fact, it does not appear that this case ever gave rise to the suspicion of arteriosclerotic mental disease (no bodily weakness until the later stages of tuberculosis, no unsteadiness, incoördination or tremor, no certain evidence of amnesia, no disturbance of speech).

If our first case be not of catatonia, it may possibly be of melancholia in the Kraepelinian sense. It may freely be granted that the case shows various phenomena found in melancholia. We ground our diagnosis on *apprehensive excitement, depressive delusions, inaccessibility, resistivism, mutism, stereotypies of posture, stereotypies of movement and speech, rhythmic movements, impulsive acts, incoherent and disconnected affectless talk, episodes.* These phenomena not only form the vast majority of those which Kraepelin describes as characteristic of late catatonia, but are also in the main not characteristic of melancholia. The possession of certain traits also found in cases of melancholia cannot be said to militate against the diagnosis late catatonia. In any case none would, we suppose, assert that either of the present cases is a typical case of melancholia.

In the above discussion, we have preferred to deal wholly with the Kraepelinian categories and naturally do not object to any critics who may choose to analyze our cases from some other point of view. Adopting the Kraepelinian point of view, we are merely seeking to follow Kraepelin's dictum that "anatomy must decide" in this group.

Turning now to a consideration of the angular gyrus and the possible results of its injury, we find the literature rather full and somewhat dubious.

The parietal area starting with the *precuneus* on the mesial brain surface, includes on the lateral brain surface the *postcentral gyrus* back of the Rolandic fissure and the (superior) *parietal gyrus* at right angles to the postcentral gyrus and running back to the parieto-occipital fissure; within the angle made by the postcentral and (superior) parietal gyri is a sub-region sometimes known as the *inferior parietal lobule,* containing from before backward the *supramarginal gyrus,* the *angular gyrus,* and the often less definite *posterior parietal gyrus.* Thus, could we trust the anatomical landmarks as affording any index or suggestion of functional differentiation, we should have to consider six constituent regions of the parietal lobe (1. postcentral, 2. superior parietal, 3. supramarginal, 4. angular, 5. posterior parietal, 6. precuneus).

If we turn to the cortex histologists, we find that Campbell (1905) differentiated cortex of but four types in this region, *viz.,* the postcentral, the intermediate postcentral, parietal, and common temporal types.[1] As to the controversy over the histological differentiation of the postcentral gyrus, we need not here concern ourselves; nor yet with Campbell's objections to Flechsig's claim of a special supra-angular portion of the superior parietal gyrus, said to ripen early (myelogenetic area 14). The parietal area of Campbell covers the precuneus, the superior parietal gyrus and the anterior part of the supramarginal gyrus.

Concerning the angular gyrus, Campbell first remarks, "The inferior boundary on this surface is the hardest of all to settle; approximately the ramus horizontalis (interparietal fissure) along with the ramus occipitalis of the intraparietal fissure form a dividing line, but these sulci certainly do not constitute a precise limit, for although it is exceedingly difficult, almost impossible, to determine the exact point where "parietal" cortex ends and "tem-

poral" begins, on account of confusion of type, yet I think it correct to say that the "parietal" type tends to cross the horizontal sulcal line and to trespass on the upper part of the angular gyrus, as well as on the upper and anterior part of the supramarginal convolution."

If this be true, then it may be conceived as possible that a few centripetal projection fibers do reach a portion of the angular gyrus, although there seems to be reason for supposing that not many such reach any portion of the parietal area of Campbell.

In so far then as the angular gyrus may partake of the nature of the parietal cortex, we may, in default of more exact knowledge, agree that it has in part a rerepresentative function with respect to sensory impressions (Hughlings Jackson's line of thought); the major portion of the angular gyrus is regarded by Campbell as belonging to the common temporal cortex as defined by him (see Campbell's diagram, p. 158). From Campbell's general adherence to the Jacksonian idea of levels, we assume that he would consider (a) postcentral, (b) intermediate postcentral, and (c) parietal to be (a) receptive, (b) representative and (c) rerepresentative.

Campbell states that with respect to a left-sided *word-hearing* center in the angular gyrus, he can find no histological evidence of differences on the two sides (p. 173). Larinow states that he has produced movements of the ears in animals by faradization of the angular gyrus (C. p. 262). Ferrier also produced lateral movements of the eye (and also of the head?) toward the opposite side by stimulation of the angular gyrus. But Sherrington and Grünbaum failed to elicit any movements (in the higher apes) by stimulation of the angular gyrus.

Ferrier thought that the angular gyrus should be included in the visual area; but it is now commonly believed that, in his experiments, he must have injured underlying optic fibers and that the angular gyrus has no visual function. However, v. Monakow (to use Campbell's words) "promulgates with some emphasis" a view "that there exists no part of the occipital cortex, and possibly none of the cortex of the angular gyrus, with which the macula lutea is unconnected." (Campbell's translation.)

"Clinical observations with subsequent autopsies cannot alone decide the question of the real extent of the human visual area, on

account of peculiarities in its blood-supply on the one hand, and on account of the possibility of new tracts being brought into operation on the other. But the evidence concerning the last-mentioned point, along with that showing that the macula remains intact even after bilaterial destruction of the occipital lobes in the narrow sense, and finally the results of the study of secondary changes, necessarily suggest that the visual area occupies, in addition to the entire cortex of the individual occipital gyri (Cuneus, Lobus Lingualis, Gyrus Descendens, Occ. 1-Occ. 3), at least the hinder part of the gyrus angularis." (Gehirnpathologie, p. 468).

Inasmuch as our first case does show lesions of the hinder part of the angular gyrus, to say nothing of apparent shrinkage of the whole occipital pole, it is interesting to note that Campbell says that " a further point of importance is that a deep lesion in the left occipital lobe seems more likely to bring psychic defects in its train than one affecting the right."

Besides psychic blindness, much has been said concerning alexia (Kussmaul's *Wortblindheit,* Dejerine's *cécité verbale pure,* Wernicke's *subcortical alexia*), that is, an inability to comprehend written or printed language, despite the fact of perfect vision of the letters. To quote Campbell once more: " Clinically, many degrees and varieties of this affection may appear, but fortunately there is almost unanimous agreement concerning its pathological anatomy. In 10 or more cases which have been carefully examined (those of Monakow, Redlich, Verrey, etc.) the surface lesion has been confined to the region of the left angular gyrus and the second occipital convolution, and usually has spread sufficiently deep into the underlying white substance to include the fasciculus longitudinalis inferior, an important band of fibres, the connections of which we shall have to mention presently. And although, in some instances, other bands, *viz.,* the fasciculus longitudinalis superior, the occipito-thalamic radiations of Gratiolet, the forceps major, and in a few cases fibres pertaining to the splenium of the corpus callosum have been involved, the stress of the injury seems always to have fallen on the band first alluded to. Also it is becoming an established doctrine that destruction of the cortex in the region of the angular gyrus by itself, or of the fasciculus longitudinalis inferior by itself, is insufficient to produce alexia; the two must go

together, and for the production of the clinical manifestation long and short systems of association fibres necessarily must be destroyed."

But, in our case, there is no evidence (so far as total brain sections stained by the Weigert myelin method show) of involvement or a destruction of these systems of association fibers.

A review of what Heilbronner has to say in his systematic summary of aphasia, apraxia, and agnosia in Lewandowski's *Handbuch*[7] adds nothing further to the above considerations. "The recognized frequency of reading-disorder with lesion of the (left) angular gyrus is explained by the interruption of communications between the occipital lobe (callosal connections with the *right* occipital lobe also considered) and the sensory speech area." Schuster seems to have shown (1909) that interruption of the *left* optic radiation as such does *not* produce alexia.

Another symptom attributed by many authors to angular gyrus lesion is *conjugate* deviation of the eyes (Landouzy and Grasset, Wernicke, Henschen). Others (Charcot and Pitres, Flechsig and von Monakow) deny this. Lewandowsky (1910) concludes that the parietal region, the inferior parietal lobule and the angular gyrus surely have something to do with conjugate deviation, although the angular gyrus localization has proved to have slight localizing value as compared with the middle frontal gyrus localization, proved by Oppenheim and Sahli in human cases (abscesses).[8]

Henschen reasserts (1910) that no portion of the optic paths and especially not the inferior longitudinal fasciculus sends fibers to the angular gyrus.[9] It would appear that our cases offer strong support to that contention.

II. Case A.

CLINICAL HISTORY.

History.—(B. S. H. 8065, Path. 1912.5). Born in Ireland; parents dead. She went to school until 17; then came to her sister in this country and began working, earning $10.00 a month as chambermaid in a hotel. For the next 10 years patient worked in three different places as chambermaid; before she married at 27, left hotel work and worked at the New England Conservatory of Music at $15.00 a month. It is stated by her sister that she was always well liked, capable, neat in dress, tidy in habits, read good

books and was religious; that she was not given to extremes in anything and had no mannerisms.

Two years after she married she gave birth to a dead child. This was a great disappointment to both her husband and herself, since they desired children. Other uterine history is that she had irregular and painful and profuse periods. Never again pregnant. The hypothesis of syphilitic infection must be here entertained (no test by Wassermann method); or perhaps due to the uterine tumor that produced menstrual disorder. The autopsy showed (besides the arteriosclerosis practically all above the diaphragm) some duropial adhesions in the cervical region, possibly due to old syphilis.

As a housewife she was successful, planned well with her money, was neat, used no alcohol, could sew and was right-handed. She worried over her husband's drinking habits, which it is stated he contracted eight years after their marriage. But for this, sister thinks the marriage would have been a happy one.

Six weeks before coming to the hospital (43 years of age) she had been caring for her sister who had broken her ankle, and one day went to church with her and *suddenly fainted in church.* After that she complained often of her head and worried about small things. Sat looking over her small possessions in her trunk and would say: "My head is sick. Go and get the priest. What'll ever I do—What'll ever I do!" Four days before going to the hospital she thought her husband was going to leave her, since he asked for the house key (to go to mass). After this she was excited and restless, often saying: "Jesus, Mary and Joseph, what will I do—get a priest for me," thinking she was going to die. During this time she did nothing and was not violent until they came to take her to the hospital, and then she screamed and was subjected to restraint.

Physicians' Certificate.—Patient said: "Don't take my John from me. They are going to kill each other. They can't arrest me—I never did anything. They can't put me away. I am going to be left here all alone tonight. Save my soul. I am going forever. Is there a just God? We will all be murdered tonight. They are going to kill John down there. I will jump out of the window." She refused a glass of water, although previously having asked for a drink of water, claiming that it might be poison. Lately she has absolutely refused to take food or medicine. About two weeks ago she claimed to be choking, but on examination everything was normal. Now we cannot go near her, as she is suspicious of everybody and it is impossible even to examine her. She was continually striving to take her clothes off and made an attempt to go out of the window. Excitable and extremely restless. She has been continually making claims that some people have been persecuting her, and she has been constantly in fear of arrest.

Physical Examination.—Emaciated and weak, probably from refusal of food. Has a rapid pulse rate, sordes on teeth and has hemorrhoids. Is constipated, urine is high colored and concentrated, but no albumen.

Her expression is anxious and apprehensive. Thinks her husband and nephew are to be killed. Believes the food is poisoned. Tube fed. For two weeks after admission she was fed by tube and remained very much confused, excited, restless most of that time, wandering about the ward, disrobing, refusing to eat or talk and moaning and muttering incoherently. She became very much exhausted, and finally, after five days, she went to sleep and slept nine hours, after which she seemed stupid and drowsy. After that she took a fair amount of nourishment and slept pretty well, but lay in bed with clothes drawn up over her head and would not talk at all. It is thought that she was not as confused or as suspicious as when she came in.

In 10 days she began eating again, began to answer questions and looked much brighter. Then suddenly became confused and excited, rushed about the ward, frightening the other patients exceedingly, and was transferred to a more disturbed ward. Peered about and started at each sound as though influenced by hallucinations. Four days later a note states that she was still confused and resistive and had to be tube fed. When in her room made an outcry most of the time. Slept poorly.

The next month's notes state that she was apprehensive and distressed. Imagined she had no blood, no bowels, that her back had been stripped off and she was being hanged by the neck. Clenched her throat until it was red and almost raw from her finger nails. When food was placed in her room and the door shut she would eat a good part of it, but would not take any from the attendants. "Worrying herself to a shadow."

A week later than this, three months after admission, a few detached sentences, such as the following can be distinguished: "Can't you see my head can't go through that wall? Sure, I am somebody, I must be somebody. Oh, don't try to do that (as she is being lifted into her bed). Can't you see there is no bed. Don't hold on to me. You will all shut yourselves in here, too. Oh, sure, I am somebody. Oh, there is no bed, there is no room," etc. Appearance of great suffering and exhaustion.

Four month later, confused, apprehensive state most of the time. Mute. Resistive. More or less destructive. Physical examination poor.

A year later.—Still untidy and destructive; sometimes unexpectedly violent. At other times she would allow other patients to strike her and pull her hair without resistance. Lay about on a bench and dozed. When awake pulled at her hair and whined and cried out. No longer any appearance of apprehensiveness.

Two years later.—No change. Untidy and at times very noisy and destructive. Masturbated. Frenzies of excitement.

In April, 1910, five years after admission.—On a chronic, noisy, untidy ward. Had quiescent periods, when she simply lay about on a bench mute, and again, became much disturbed and cried, or rather roared most of the time, and wandered about in a staggering fashion, stamping her feet and seeking somebody to attack. Would fasten her hands in a patient's hair while she was sitting quietly, with a grasp like iron, in spite of her feeble appearance. Resistive about going to bed. Not taken out of doors

except in summer, as she would lie down in the path and refuse to walk. At times she shouted and yelled the same phrases over and over in a high-pitched, monotonous voice for hours at a time. She resisted passive movements.

1911.—Noisy, shouting over and over incoherent sentences and stereotyped phrases. Still very untidy.

June, 1911.—Remained in bed in the same untidy, noisy state. Some days she would shout most of the day, using unintelligible phrases in a high-pitched, monotonous tone. Noisy at night. Small doses of sedatives.

December, 1911.—Six years after admission. Failing physically and, though she resisted physical examination, it was discovered that she has a marked dulness over posterior lobe of both lungs, especially the right, with moist rales. No expectoration.

January, 1912.—Gradually became weaker and died.

Diagnosis on admission, involution melancholia; diagnosis on discharge, dementia præcox.

POST MORTEM EXAMINATION.

Autopsy 13 hours post mortem (M. M. C.).

Cause of Death.—Pulmonary tuberculosis (cultures from).

Acute or Active Lesions.—Cystitis; sacral decubitus; hemorrhage into left ovary; hemorrhage into left renal pelvis and into cyst of left kidney; internal hemorrhagic pachymeningitis with partial organization.

Chronic Lesions.—Emaciation; aortic, coronary, internal mammary, splenic arteriosclerosis; slight hypertrophy of heart; slight atrophy of liver; slight atrophy of thenar muscles; fibromyoma of uterine fundus (6 cm. in diameter) with underlying chronic endometritis; right ovary cystic; chronic focal adhesive peritonitis (ectum, left Fallopian tube, broad ligament, and ovary involved in adhesions; no lesion inside rectum to correspond,) possibly healed syphilis; mammary atrophy.

Anomalies.—Right pupil smaller than left; asymmetry of sternum (lower end deflected to right); left clavicle depressed; nose deflected to left; rigor mortis absent in right arm, trunk, and neck (13 hours post mortem); contractures (?) of hands.

Nervous System.—In addition to slight internal hemorrhagic pachymeningitis, very slight calvarial adhesions, calvarial depressions to accommodate arachnoidal villi and middle meningeal arteries, comparatively thin temporal bones. There are lesions as follows:

Variations in Consistence of Encephalon.—Olives, occipital and frontal poles firmer than hippocampal gyri and cerebellum; the latter in turn firmer than the temporal cortex (this suggests topographical variations in degree of gliosis).

Slight Atrophy of Encephalon.—Brain weight 1145 gm. (Tigges' formula would yield 8 x 1 body length 150 cm. = 1200 gm.).

Cyst of softening of grey matter of left angular gyrus (of almond shape, major axis nearly at right angles to longitudinal fissure, external extremity

slightly posterior, about 4 x 2 cm.) with overlying *sclerosis of pial vessels, notably veins. Slight retraction of left occipital tissues* (atrophy?). *Edema of Gasserian ganglia* (pituitary firm). *Adhesions of dura to pia in cervical region.* Slight basal arteriosclerosis (no patches or yellowing).

Frontal sections through the two hemispheres in the plane of the focal lesion were stained by the Wiegert and Wiegert-Pal methods for myelin and by Mallory's anilin blue method for connective tissue. Smaller blocks from the injured, adjacent, and coördinate areas were stained by various methods to arrive at some notion of finer details.

Large brain sections through the middle of the lesion pass through planes posterior to Dejerine's frontal section No. 137 (Dejerine's fig. 266), and display relatively intact (beginning at a point superior to the lesion and skirting the left hemisphere) the *first occipital gyrus, superior parietal gyrus, cuneus, lingual gyrus, third occipital gyrus,* and *second occipital gyrus.* Indeed, small limiting portions of the angular gyrus are likewise intact, namely portions adjacent to the interparietal sulcus above and to the second occipital gyrus below. The plane of section also demonstrates some intact white matter apparently isolated from the underlying white matter: this appearance is explained by the shape of the lesion which is not quite round.

The process in and about the focal lesion seems entirely chronic or (with due respect to the neuroglia appearances) perhaps very slowly progressive. Of first importance is the histology of the cyst itself. The cyst is evidently due to an old necrosis of tissue supplied by a terminal branch of the parieto-temporal branch of the sylvian artery. The thrombotic arterial branch itself was cut at a point about 1 cm. posterior to the anterior border of the cyst of softening and can be seen making off from a previous blood-filled artery. The shrinkage of tissues due to the cyst has caused considerable buckling of the thrombosed artery, unless we are to suppose that the disease process has lengthened the artery. The thrombosis is of ancient date, as evidenced by the canalization of the connective tissue contents of the vessel. Mallory's anilin blue connective tissue stain shows a somewhat denser portion of small dimensions in the middle of the obliterated lumen: it is possible that this represents the remains of an old compressed strand of dissected-off endothelium. If this hypothesis is correct, it is possible that the original thrombosing process was like that found in various acute meningitic processes (pneumococcus and typhoid meningitis, for example). The sections show that the thrombosis extended for at least 1.5 cm. probably a greater distance (in the total-brain section block this vessel can be followed as a gray cord at the bottom of a sulcus stretching forward, outward, and slightly downward to communicate with the main parieto-temporal branch).

The cyst itself shows some collapsing of its numerous connective tissue-septa. Between these septa, which are rarely thicker than pulmonary alveolar walls, are collections of large phagocytic cells stuffed with degeneration products, but with nuclei (not as a rule pressed strongly to one side) rather quiescent-looking and globular. There are numerous small

blood-filled vessels running through the connective tissue septa. There are next to no polynuclear leucocytes in the cyst spaces. There appear to be few lymphocytes and fewer plasma cells (if any); nor are there any accumulations of mononuclear cells about vessels at any point. There is considerable edema in places, and a large vein next to the plugged artery is filled with coagulated albumen.

The edges of the cystic spaces are remarkably definite and only slightly exhibit any suggestion of edema. The neuroglia cells are often supplied with cell-bodies of comparatively large size; but the nuclei even of such cells are not often vesicular. Still, it cannot be denied that the larger size of the neuroglia cell-bodies gives something the aspect of an active zone.

As one passes back from the cyst edge, there is evidence of some loss of tissue, since the small vessels lie in vacuoles containing considerable coagulated albumen, indicating ante mortem dilatation of these spaces. It seems clear that fairly numerous nerve fibers have been lost from the white matter surrounding the actual cyst. There are relatively too numerous capillaries in the tissue of both cortex and white matter surrounding the cystic spaces; and this excess of capillaries shades off gradually in less than 1 mm. into tissue supplied with a normal number of capillaries.

The pial edges of the cystic spaces are interesting from the slight undermining of the tissues which makes it perfectly clear that the subpial zone has great vitality as compared with the underlying layers. This is probably to a great extent due to a separate blood-supply by short meningeal vessels. But there are also signs that the neuroglia itself in the subpial zone is capable of strong reaction: some sections show that the inner face of the subpial layer, as it presents upon the cyst, is very markedly beset with neuroglia cells having expanded cell-bodies. In some places the subpial zone, as it overhangs the cystic space, has become a fifth to a fourth thicker than in adjacent regions where it overlies relatively normal nerve-tissue.

There are also ample opportunities for studying the differential vitality of the various cell-layers.

III. Case B.

J. G., No. 9599. Autopsy No. 1913-47.

Family History.—Patient's father was peculiar and at present (1910) has chronic melancholia. Mother is very nervous, and one sister is in a sanitarium for nervous breakdown.

Personal History.—Patient went to the Boston schools until he was 14. Went into insurance business at 18. He was very successful and remained with one company, A, for 18 years, when he was discharged because his commissions did not show a sufficient increase. This misfortune was a great shock to him and he worried excessively about it, but he at once secured a position with Company B, did well with them and remained there 18 months, when Company A offered to take him back and he

accepted. He was given a very bad district in the slums of Boston, had trouble with his agents and all went wrong. He was admitted to the McLean Hospital February 1, 1910, with the above history, and the onset was given as 12 days before admission, when wife came home from the theatre and found him talking incoherently to himself, which condition has persisted. He has made several attempts to get away from the house and on the morning of admission to the hospital tried to jump from the third story window.

Physical Examination.—On admission to McLean was negative. All his answers to questions were given with more or less hesitation and with many incomplete sentences. He did not appear particularly depressed. There was no evidence of motor retardation. The immediate data of his experiences he took in but did not reason about them. A good deal of thinking disorder. Five days after admission, while sitting in chair, he suddenly slumped to the floor. Reflexes not disturbed. For 24 hours he lay in bed apparently dazed, keeping his gaze fixed very steadily on any new point of attraction. He would not eat and for four days he was tube fed, and at the end of the fourth day again gradually relapsed into the same dazed condition. Pulse 85, temperature in axilla 100. He would open his eyes after several commands, but had to be catheterized. In eight days he became a little more restless, temperature rose to 105, pulse 120, respirations 104. Two hours later, temperature dropped to 101, pulse 110, and the temperature became normal the same evening. Ten days after the initial attack, he suddenly made a rush for the window and resisted being put back to bed. Some days he had periods when he seemed amused; then would suddenly become tense and surly. Between the 15th and the date of admission to this hospital, the 23d of April, he has been much more inclined to fall into fixed attitudes, staring out of the window for 30 minutes at a time without perceptible change in position. Has acquired many habits, such as washing hands a certain way, standing in one place in the room, etc. Often complains that his head feels as if it were stuffed with mud. Often gives answers that are superficially correct, but does not want to take the trouble to put any real thought on anything he says. The striking things of his history are the *heredity,* the *efficiency* up to the age of 36, and a *sudden slump* which was recognized by his employers; followed in two years by marked physical change and increase in his mental incapacity. When he came to this hospital he was 38. His habits were said to be good. Commitment papers indicated that he was *suicidal;* and the patient said detectives were on his track, that he was going to carry a pistol in each pocket to protect himself, that a lodger across the street was a detective and on the watch for him, that there was a scheme in the insurance company to drop him out of sight. Patient was pugnacious and agitated, attempted to jump out third story window.

Other Facts.—Has failed mentally for some months; has lost his position with the insurance company. Has frequently spoken of revolvers and attempted to procure them. Has attacked his wife.

Physical Examination.—Shows a high narrow palate. Sluggish patellar reflex and *unsteady gait.* Coarse tremors of the tongue, disconnected speech—otherwise is negative. He talks constantly, twists his face into many peculiar expressions, but there is no speech defect. *Consciousness* is said to be greatly diminished. Evidently has *hallucinations* of sight and hearing and his attention is markedly blunted. His *memory* is poor and his *orientation* also. His train of thought shows a remarkable flight, of which the following is an example: "My head was driven right down between my shoulders. I was the littlest Jesus in the manger, started in eating straw. If I had been a horse, I'd have eaten my old black hoof. I have palpitation of the heart and liver at seven cents per pound. My father tried to shoot himself in Ayer Junction and Groton, Groton, Groton oil, capital G-R-O-T-O-N spells Groton, rotten whiskey is 15 cents a bottle in Springfield. Bancroft drank vinegar to quench his thirst. There's your knuckle, you bite it and its pinochle. Look at the Spanish-fly blister (pointing to a part of his body where there is no mark of any kind). Of course, suicides run in families, settled in Maine and moved up." In *May, 1910,* is reported as being disturbed, and destroyed clothing. In a highly excited condition, talking continually in a loud, rapid manner and going through many peculiar, purposeless movements. Treated with warm baths and became more quiet, which continued to the last of July, when he had a *temperature* of 103, which persisted for about a week and then became *normal.* In November of the same year, the note mentions that he is in a dull, stupid condition, cannot answer questions intelligently, and seems to be very much *demented.* Owing to his inactivity, his feet are swelling; he has become extremely untidy in his habits and does all sorts of repulsive things. For example—plugging his nose and ears with feces and rubbing feces over his body and hair. In *May, 1911,* he is somewhat untidy but appears brighter, and the next December he is noted as taking more interest in his surroundings. Has been employed in the industrial room and picked up considerably. Enjoyed going to the entertainments, but is said to be semi-catatonic. Later, leaves arms in position placed by examiner for a long time, and was at this time presented at staff meeting where four people thought him *dementia præcox,* though "manic" and general paresis were considered. *Two years* after the initial attack, he is reported as being stupid and answering questions in a whisper, sometimes intelligently, other times irrelevantly and was found to have a temperature of 99 degrees; and had an attack of *otitis media* in the right ear which persisted for 10 days with a temperature range of *98 to 104.* He was at that time tested by the Von Pirquet, which was *negative.* From then on there is a record of his temperature during the remainder of his stay in the hospital which is a continuous one, sometimes for weeks ranging decidedly above normal with upshoots to 102, and pulse between 80 and 118; and during the spring of 1912, the highest temperature not reaching above 102, the second Von Pirquet in March being negative, there appeared a small fluctuant tumor on the left back, between the sixth and tenth ribs near the spine, and tubercle bacilli were present. From then on the notes talk

of discharges from "cold abscesses" in various parts of his body, and physical failure; incoherence in conversation.

August 15, 1913.—Edema of the legs and irregular pulse was noted, which increased with shifting dullness in flanks and a distended abdomen. Physical signs of general tuberculosis and edema.

Summary.—A man, in hospital 44 months and three days, died at the age of 41, after an illness which first appeared to be manic depressive; diagnosis later was dementia præcox with an onset at the age of 36. Commitment at the age of 38 with various attacks of unexplained temperature, and finally died after a long drawn out physical illness, with multiple discharging points of tuberculous abscesses.

The points in favor of general paresis were that at the age of 36 there was a period when business fell off, followed by a period of excitement and a slump, followed by demented condition. There was a tremor of tongue, slight unsteadiness of gait, and the *destructiveness* and repulsive *untidiness* mentioned. But there were no physical signs, and cell-count negative. Consistent with manic depressive phychosis, were suicidal tendencies, heredity, flight of ideas, a reason for worry (being discharged). For dementia præcox—he was discharged because of *inefficiency* at the age of 36—he was *incoherent* in his talk and *superficial* in thought. There were evidently imperative *impulses* when he would try to jump out of the window. There was a period of *mutism* and *refusal of food* and a *stupor* in which he had to be catheterized. There was the assumption of *fixed attitudes and mannerisms,* and lack of ability to think coherently. He thought that the *insurance company was against him,* and he was evidently *hallucinated* for sight and hearing and his speech was rapid and incoherent. His early dementia and inactivity and semi-catatonic reaction make a diagnosis of dementia præcox most probable.

The autopsy showed visceral and brain lesions of extraordinary interest. There was a general anasarca. There were areas of decubitus and there were linear surgical wounds, some still discharging, on the inner thigh, one at Poupart's ligament, one near the anterior superior spine of the ileum, one on the external side of left thigh, and one left posterior inner outcurve of the eighth rib, and there was a swelling 5 cm. in diameter over the sternum at the junction of the xiphoid process. There was also an ascites of a chyliform nature which is unusual.[1]

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[1] Battey-Shaw Journal of Pathology and Bacteriology, volume 6—1900, page 339, notes that Bussey in 1889 first classified various reported cases of chyliform effusions of which there were 115 cases, and 54 cases of hydrothorax of a chyliform form-nature and two of chylus pericardium, and that these were usually due to rupture of or pressure upon the thoracic duct. Allbutt in the system of medicine, page 515, gives the mechanism of production of chyliform effusions as:

1. Failure of heart as a pump, falling of arterial pressure, rise of venous pressure near the heart, followed by fall in capillary pressure and absorption of fluid from the intestines and a diminished urine.

There was in this case no evidence of pressure on the thoracic duct by lymph nodes or growths of any sort, but the heart entered into the general tuberculous process by supporting an abscess which was continuous with the swelling over the xiphoid process that infiltrated the eight intercostal muscles on each side internally, through the articulation of the eighth rib on the left to the anterior mediastinal tissues and to the pericardium and epicardium over the apex. Whether this incapacitating of the heart at the left apex and the surrounding pericarditis was *the* cause of the general anasarca is, of course, an hypothesis, but since all the tissues were flooded with fluid, it is not impossible to suspect that reversed venous pressure and gravity would permit and encourage the transudation of the chyle through the receptaculum chyli.

Tuberculous processes were also in the lungs, in the kidneys and in the left psoas muscle, and there was in the brain a focal area of firmness in the anterior portion of the left angular gyrus, which upon examination proved to be a fibroid tubercle, without caseation. The remainder of the brain was unusually plump and normal in appearance.

We have in this case then an unusual combination of visceral tuberculosis, showing a rare variety of *ascites, a psychosis appearing at the age of 36, and an irritative process* limited to the grey matter in one area of the brain cortex in the post-Rolandic region.

H. P. M. 18. Age 41. Head, Trunk and Cord: M. M. Canavan.

Body of a fairly well-built and poorly nourished white male, 172 cm. in length. *Skin* waxy white, slightly discolored in patches in the neck region. No lymph nodes palpable but skin is full to *saturation* of fluid in all extremities, neck, arms, hands, scrotum and feet, and the *abdomen* protrudes and nearly obliterates the umbilicus.

Pupils.—Right, 0.7 cm., left, 0.8 cm. Rigor mortis present except in arms; *contractures* of legs to point of 45°; *body* in right lateral decubitus. *Decubitus* (superficial) over sacrum 4 cm. in length. There are several *surgical wounds* (linear), 2-3 cm. in length, some of which have healed, some still discharging; they are located as follows: two on right *inner thigh* in upper third internal to sartorious muscle; one at *Poupart's* ligament 4 cm. internal to left anterior superior spine of the ileum, one on external side *left thigh* 10 cm. below trochanter major, and one on *left chest* posterior near the outcurve of the eighth rib. There is a *swelling* 5 cm. in diameter over the sternum at the junction of the xiphoid process. Penis not examined. Tibiæ smooth.

Ventral Section.—Panniculus nil—skin and muscle very thin over thorax and abdomen—the latter particularly, and the stomach and coils of white

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2. Hydraemic plethora rise of main pressure throughout vascular system.

3. Stage of dropsy caused by high capillary pressure and increased permeability due to malnutrition.

4. Hydraemic plethora leads to ever increasing over-filling of the heart cavities, and to failure of the heart.

intestine well out of the incision, as does quantities of opaque thin fluid, (several thousand cc.). Spleen free and large—liver floats away from right side and from diaphragm. Appendix 6 cm. in length. Peritoneum looks clear and also the intestines except for a short space of the small gut where the visceral peritoneum is grey and parboiled in appearance and shows no miliary tubercles. Mesenteric lymph nodes slightly enlarged; fat in mesentery infiltrated by fluid. Diaphragm not measured. Prolapse of rectum slight.

Thorax.—Making the cuts through the skin the swelling before mentioned is undermined and at once a quantity of yellow-green pus exudes. Stained specimen shows detritic remains of cells and tubercle bacilli. This abscess has ramifications of an unusual extent, for connected with this local well of pus are other pockets infiltrating the eighth intercostal muscles on both sides and extending laterally to a line dropped from the nipple on the left to the mid-axillary line on the right, and internally through the articulation of the eighth rib on the left to the anterior mediastural tissues and to the pericardium and epicardium over the apex. *Lymph nodes* in the area outlined are enlarged and all tissues are edematous.

The pleuric cavities are filled with fluid, the lungs floating in it, and the left lung is attached to the chest wall at the apex and to the posterior wall at the base; the right lung adherent at base and diaphragmatic surface. Peribronchial lymph nodes enlarged.

Heart.—Weight (with pericardium and much edematous tissue surrounding), 410 gm. It is impossible to separate the peri- from the epicardium except by dissection, and the apex of this spreading puriform abscess is discovered to be in the epicardium over the left ventricle near the base of the heart, and extends also laterally so that cross-sections of the muscle reveal areas of cheesy material following the coronary left (descending branch) and also in the edematous epicardium. At the point where the pus is the most abundant (near branching of the left coronary), the heart muscle otherwise firm, is semi-disintegrated and softened and of a lighter color. Measurements:

T. V. 11.0 cm.	P. V. 7.5 cm.	L. V. 1.0 cm.
M. V. 10.0 cm.	A. V. 8.0 cm.	R. V. 0.4 cm.

Endocardium slightly grey. Right auricle very small and occupied by a thick cruor clot. The tricuspid valve is also adherent at its otherwise free edges. Note—(Condition of heart perhaps responsible for chylous ascites?).

Lungs.—Combined weight, 380 gm. Lungs are almost collapsed, left shows miliary dots on pleuric surface and a slight tuberculous process at apex (no cavitation) with an enlarged node clinging to apex wall. Cut section shows points of pus in some bronchi with reacting peribronchial infiltration. The right lung shows congestion but no tubercles and a cheesy spot on the lowest lobe posterior, which when separated from the thorax, leaves an infiltrated chest wall near the spinal column. The left upper lobe leaves the same appearance to the parietal pleura. On the right

eighth rib posterior near the vertebra is a ragged tear in the parietal pleura and an oozing pus point size of a walnut.

Organs of Neck.—Not removed.

Abdomen.—Spleen. Weight, 250 gm.. Capsule not thickened; organ very plump. No malpighian bodies; trabeculæ indistinct, pulp rich in amount but not soft, no apparent edema.

Adrenals.—Plump and yellow; centrally softened.

Kidneys.—300 gm. Perirenal fat, stringy and edematous. The fibrous capsule not thickened. Cortex, which measures 0.6 cm., swells slightly over the capsule. Marked differentiation exists between medulla and cortex, the first very brilliantly red, the second most brilliantly yellow of an ochre shade. Examining this more closely the yellow has indefinite-edged, tiny, white linear specks very close together throughout the cortex. Blood-vessels in the cortex brilliantly outlined by vivid red color. Calices and pelves negative.

Liver.—880 gm. The entire surface has a contracted appearance, though the capsule is not thickened, and on section shows not more than a usual amount of resistance. No increase of interstitial tissue made out—liver quite bloody. Gall bladder pale and wall thick; contains no stones.

Pancreas.—Not examined.

Gastro-Intestinal Tract.—Stomach very large and distended with gas; rugæ present, mucous wall fairly normal in appearance, pylorus free. No change in mucous membrane at any point of intestine; the section of that point which showed change on serous coat gave no corresponding change inside the gut.

Genito-Urinary Tract.—Bladder large and free, prostrate not enlarged. Testes not examined.

Special Examination.—Pushing a probe through the areas mentioned under general description on right inner thigh the free end of the probe ascends on the inner side of the sartorious muscle and is visible under Poupart's ligament. The right psoas muscle is thin and flabby and soft, and on cutting is seen to be greenish, brown and friable, and the fibers easily separate longitudinally, leaving individual fibers free with large spaces between, and the lumbar nerves are isolated and exposed; the neurolemnia of the nerves looks brownish (note—looks like the residual of a drained abscess). With the organs of the trunk removed, the peritoneum made as dry as may be, though every mesh of tissue has been as edematous as possible, the anterior portion of the spinal column is inspected for twists, curves or caries; none are found. The inner head of the left psoas shows pus in its substance and probably accounts for the pointing of an abscess in left groin. The lymph nodes along the spinal column are enlarged, the sympathetic chain appears smaller in caliber than usual, the retroperitoneal tissues immediately above and below the crura of the diaphragm look ragged, infiltrated and edematous.

Head.—Hair brown, mixed with grey. Scalp not remarkable. Calvarium measures—frontal 0.6 cm., temporal 0.4 cm., occipital 0.3 cm. Dura not thickened nor adherent, pia slightly translucent but at no point opaque.

Some excess of cerebrospinal fluid. Brain softened except for occipital tips and one small focus 1 cm. in the most anterior portion of the left angular gyrus. Basal vessels clear: pituitary small. Ganglions and middle ears negative. Brain weight, 1450 gm.

Cord.—Another attempt to locate if possible any caries of the vertebrae from the posterior aspect, and no curves nor dislocations nor softenings seen, though each vertebra was inspected. Some slight rotation of right fifth and sixth rib on the right. Back muscles very edematous. Transverse processes narrow.

Anatomical Diagnosis (1913.47).—Poorly nourished; general anasarca; unequal pupils; contractures; decubitus, sacral; surgical wounds, legs and back; abscess over sternum (tuberculosis); chylous ascites; mesenteric mediastinal and bronchial lymphnoditis; prolapse of rectum; hydrothorax; chronic obliterative pleuritis; tuberculous pericarditis; pulmonary tuberculosis; intercostal tuberculous abscess; tuberculosis of kidneys (?); beginning cirrhosis of liver; destruction right psoas muscle; pus in left psoas muscle; cerebral malacia; focal *sclerosis* left angular gyrus; brain weight, 1450 gm.

Miscroscopic Examination.—As in the previous case, the miscroscopic examination has been for the present confined to local conditions about the tubercle, except that no total brain sections have as yet been prepared. The tubercle bacillus was demonstrated in section. There was a moderate number of giant cells in the wall of the tubercle both on the side facing the pia mater and on the internal aspect. A bacillus was occasionally demonstrated in a giant cell. No other organisms appear to be found in the lesion.

It would, of course, be particularly important to know the age of the tubercle. So far as indications go, the tubercle may have been a very old one. The quasi-capsule of the tubercle was from 1 to 2 mm. thick and appears to be somewhat thicker on the pial side than elsewhere. In the outer portion of the fibrous capsule there are a moderate number of vessels whose sheaths are infiltrated with lymphocytes and a moderate number of plasma cells. No endothelial lesions were found. Outside the fibrous capsule there are some small vascular twigs with a slight infiltrate in their walls; but within far less than a millimeter from the capsule, infiltrated vessels were no longer in evidence. Just outside the fibrous capsule the nerve cells of all layers appear abnormal, suggesting pigmentation and a kind of spongy transformation in which the nuclei are not infrequently absent. These dead or dying nerve cells are not particularly prone to show satellite cells. Beyond the range of the dead or dying nerve cells, however, the nerve cells are very generally supplied with a moderate excess of satellite cells rarely more than three to five in number. These satellite cells do not especially occur at the bases of the nerve cells but preserve a somewhat irreguar relation thereto.

Throughout the zone lying outside the fibrous capsule are moderately numerous neuroglia cells having expanded homogeneous-looking cell-bodies in which by appropriate stains ependymal dots can be demonstrated. These

expanded neuroglia cells have as a rule but one nucleus and rarely more than two. The zone of the "active" or expanded neuroglia cells passes beyond the zone of dead or dying nerve cells, and beyond the surrounding zone of nerve cells showing satellitosis, and these expanded neuroglia cells are found in otherwise normal-looking nerve tissue.

It is a question how these cells may be interpreted as to the part they play in the lesion. They do not appear more numerous or of a different appearance in the concentric zones above mentioned. They must very probably correspond to the destruction or partial destruction of nerve elements or portions of nerve elements.

A speculation could easily run to the effect that as the necrosis in the tubercle advanced, the surrounding zones themselves gradually advanced, with the neuroglia cells always in the lead. As the lesion advances, it may be supposed that the first thing which happens is the death of neurones or parts of neurones outside the zone of satellitosis in that region which we have described as looking normal but containing expanded neuroglia cells. As the destruction of elements in this part becomes more pronounced, the satellitosis enters. As death finally overtakes the cells, they lose their satellites, and hence the inner zone just adjacent to the capsule is developed, in which dead cells are seen but satellite cells are not prominent.

Meantime, whether from their superior vitality or from the fact that they can get on under less favorable circumstances, the neuroglia cells remain in comparative abundance throughout these zones and in approximately equal parts in all zones.

Accordingly, the tubercle, whatever its age, seems to be manifesting an unusually even extension as indicated both by the regularity and homogeneity of the capsular and pericapsular zones, but also by the evenly diminishing exudate about the vessels in these successive zones.

As to the reaction of the cell layers to the tubercle, no special study of these reactions has been made for the present communication. In the superficial orienting study no indications of a differential reaction were to be found. From the greater thickness of the capsule on the pial side and adjacent portions of the lesion, it might be inferred (though with no great assurance) that the region of greatest activity in the lesion was in this neighborhood and that accordingly the outer cell layers would be more particularly affected at first than the inner layers.

IV. Summary.

In the nature of things no proof can yet be offered of the genetic relationship of lesions of the left angular gyrus and catatonia. It has been claimed by one of the writers that the catatonia of dementia præcox is more a property of parietal (post-Rolandic) lesions than of anterior lesions; and the dementia præcox lesions are not coarse lacunar lesions. Little or no evidence has been

hitherto available as to the relation of *coarse* destructive brain lesions to catatoniform syndromes in diseases other than dementia præcox. It is sometimes stated that deep *left-sided* occipital lobe lesions are more often attended by mental symptoms than identical lesions of the right side. And, of course, the classical relation of the peculiar symptom *alexia* to the *left* angular gyrus (to be sure it is now alleged that the underlying fasciculus longitudinalis inferior must be simultaneously affected to produce alexia) may be urged as pointing *leftwards* to a region of greater psychic interest.

However this may be, the writers present two cases, in themselves interesting, which suggested and obtained the diagnosis dementia præcox, although the cyst of softening in one and the solitary tubercle in the other may well be regarded as withdrawing the cases altogether from the dementia præcox group and settling them in a group of nondescript, coarsely organic, and destructive brain lesion cases.

The experience of these two cases suggests further work to secure psychopathic correlates in other cases (a study of the Massachusetts material of angular gyrus material is far advanced towards completion), and to learn from a large group of so-called "late catatonias" whether there are any which really belong in the true dementia præcox group, as we are coming to conceive it.

Of the two cases, A was female, B male. A's symptoms began out of a clear sky with a fainting spell at 43. B's symptoms began with business inefficiency at 36. A died at 50, having shown no sign of arteriosclerosis after the initial faint. B died at 41, having developed severe generalized tuberculosis, but no focal nerve signs from his solitary tubercle. The diagnosis in each case was perhaps not entirely clear, especially viewed in post mortem light; but each received the diagnosis dementia præcox. There is a suspicion (but only a suspicion) that A may have been syphilitic.

Both patients showed at one time or other *hallucinations of hearing* (B also of sight), *disorder of consciousness, confusion, incoherence, mutism, refusal of food* (tube-feeding), *impulsivity, delusions of persecution, apprehensiveness* (B also suicidal), *fixed attitudes, mannerisms, somatic delusions, destructiveness, violence, stuporous states* (catatonic).

The somatic delusions were partly cephalic, and may perhaps be taken more as illusions:[10] A., *"head sick," "back stripped off and*

being hanged by neck," "my head can't go through that wall"; B., *"head feels as if stuffed with mud," "my head was driven right down between my shoulders."*

As to *alexia,* neither case could be tested.

As to *conjugate or other eye movements,* A showed nothing which was independent of other paraboulic acts, but B's eyes would sometimes be fixated for long periods upon a succession of different points in space (half an hour at a time).

As to *automatism* and *cerea flexibilitas,* A was at times (but rarely) passive to any form of attack, B showed automatism and possibly *cerea.*

As to *schizophrenia,* A ran to the autism of delusions of negation (*"Sure I am somebody, I must be somebody * * * Can't you see there is no bed * * * Oh! there is no bed, there is no room."*), B to grotesque shifting of ideas with some maniacal plays on words woven in (*"There's your knuckle, you bite it and it's pinochle. Look at the Spanish-fly blister"*).

We may now compare the cases with respect to disorders of senses and intellect on the one hand and emotions and the will on the other.

The senses in both cases appear to have been normal.

Aside from periods of *confusion* and *incoherence, delusions* of *persecution* and *somatic delusions,* the patients showed little or no intellectual disorder. To be sure, both, as stated above, showed at one time or another, *disorder of consciousness* and *stuporous states* of a catatonic appearance, and it is possible that the *hallucinations of hearing* in both cases may be explained by some sensory or intellectual disorder of an unknown nature.

It does not appear that Case A showed a special intellectual disorder before the onset of her disease; it may be thought that Case B had experienced some intellectual deterioration as evidenced by his loss of business capacity.

Concerning emotions, both patients showed *impulsivity* which was regarded, however, as not of emotional origin. Both patients were at times *apprehensive* (B also suicidal) and both appeared to have reacted as it were normally to their hallucinations. *Refusal of food* in both cases may be regarded as possibly of emotional origin, although the general interpretation of the cases seemed to indicate that this symptom was of a catatonic nature.

Both patients had at times to be *tube fed.*

As to the general emotional state, it does not appear that either patient was ever euphoric, although B had periods of apparent amusement, in the midst of which he would suddenly become tense and surly (A also had sudden attacks of excitement, but it does not appear that they were related to a particular emotional state).

As to the will, the conduct of both patients was variable. Neither was successfully gotten to work, although each was for a good part of the time not in bed.

Both patients were *untidy* from time to time.

Both patients showed a number of anti-environmental symptoms such as *destructiveness, violence* and often dangerous *impulsivity, mutism* and the like.

Little can be said regarding the sex life in either case (masturbation in Case A, occasional remarks of a sexual content in Case B).

The bodily state of patient B fluctuated from time to time in response to the tuberculosis. Patient A showed no especial physical disorder except emaciation; this was also patient B's condition.

There appears to have been no clinical evidence pointing to the moderate atrophy of the liver in Case A, or to an early cirrhosis in Case B.

Both patients suffered from pulmonary tuberculosis, and Case B showed a variety of tuberculous lesions in many organs, as well as the chylous ascites considered in detail above.

The total duration of symptoms was in Case A, seven years, in case B, five years.

It would naturally be very important to know the age of the cyst of softening in A and of the tubercle in Case B. The appearance of both lesions is consistent with their being very old, at least as old as were the symptoms; nevertheless, it may be that both lesions were either older or younger than would be indicated by the age at which symptoms developed.

The argument is fairly strong in Case A that the lesion was probably correlated with the only *fainting spell* which the patient appears ever to have shown. It will be remembered that in Case A there were no other arteriosclerotic symptoms or any other suspicion of cerebral arteriosclerosis.

With respect to Case B, microscopical analysis seems to show that the lesion may have been in some sense slowly progressive, that is to say, that the necrotic center of the tubercle may have grown slowly larger with an even and progressive expansion of the capsule, parts of which may have been destroyed in the globar spread of the process. Interesting details as to the special features of the successive zones of the lesion from within outward are given above.

If one were inclined to press analogies between these cases and the histological features of ordinary dementia præcox, one might be interested in the observation that the subpial zones in both cases, being origins of greater vitality (perhaps owing to their vascular supply), are the regions of greatest activity. Case A, for example, showed an intense neuroglia reaction in the overhanging subpial zone, and Case B also showed that the outer cell layers would be far more likely to be affected early than would be the inner layers.

This question is not one which can be resolved with such material as is afforded by these cases, nor do we know enough about the differential reactions of the suprastellate and infrastellate zones to make discussion at this time profitable. It may be noted, however, that in the later work of the Munich school, attention has been drawn to the suprastellate layers rather than to the infrastellate layers as a site of lesions in dementia præcox.[5] To be sure, Alzheimer had, so far back as 1897, noted gliosis of the lower layers of the cortex in catatonia;[11] but it appears that later work has tended to reverse this conclusion, or, at any rate, to show that the suprastellate zone is more markedly affected in most cases; at all events, this appears to be the situation in these two cases. What we now need, accordingly, is differential study of the angular gyri in a considerable series of dementia præcox cases, together with a sufficient control series.

Whether the lesions had a direct mechanical effect upon the tissues to give rise to the symptoms in these cases must remain obscure. That the gliosis with its contractile tendencies was more marked in the suprastellate region than in the infrastellate region, is merely an interesting fact that may be more a matter of coincidence than a fact of genetic value.

In work on epilepsy it was formerly claimed that very possibly the contraction of the proliferated neuroglia tissue might evoke or liberate convulsions, whereupon the simplification of neighboring tissues, structurally proved to exist before, would favor the propagation of the epileptic discharge.[12]

Much, of course, must depend upon the site of the lesions and the original function of the cells and tissue in question. Convulsions were not produced in these cases, nor is there any especial evidence in the literature that convulsions are produced by lesions in either angular gyrus.

We leave the question unanswered accordingly, from the data of these two analogous cases, that catatonia or catatoniform symptoms may occasionally be mechanical in origin.

V. Conclusions.

The writers present two cases of chronic lesion of the left angular gyrus which received the clinical diagnosis of dementia præcox. One case showed a cyst of softening and the other a solitary tubercle. It appears that both lesions may well be of suitable age to correspond with the date of onset of the symptoms. Although not in all respects typical, the diagnosis of dementia Præcox seems to have been accepted by the Boston State Hospital officers in charge of the cases. Decidedly atypical is the age of onset of the first case, at 41; the second case had its onset at 36.

The writers are especially interested in the fact that the isolated lesions in these cases are in the parietal region, a region which has been stated in previous work from this laboratory to be correlated with catatonic symptoms. Plates are presented showing the site of the lesions.

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DESCRIPTION OF FIGURES.

Fig 1, Case A.

Total brain sections of the two hemispheres stained by the Weigert-Pal myelin method.

Female, aged 50.

Cyst of softening of left angular gyrus of arteriosclerotic origin due to thrombosis of vessels (demonstrated in special sections).

There is, internal to the cortex lesion, an area of partial destruction of tissue, which is in part due to artefact, but may in part represent a mild degeneration of myelinated fibers. Traces of a similar process are to be seen internal to the calcarine fissure.

Careful external photography and dissection have so far not revealed other arteriosclerotic lesions or lesions of any other nature in the nervous system of this case.

Fig. 2, Case B.

Male, aged 41, with onset of symptoms at 36.

Photograph of the posterior aspect of the surface of section of the brain cut frontally in the region of the angular gyrus. (The photograph was made of the tissue anterior to the plane of section.)

The tubercle is a trifle over 1 cm. in a plane parallel with the convolutional surface, and is about ¾ cm. deep when measured from the pial surface to the inferior edge of the capsule.

No other lesions have been found elsewhere in the brain.

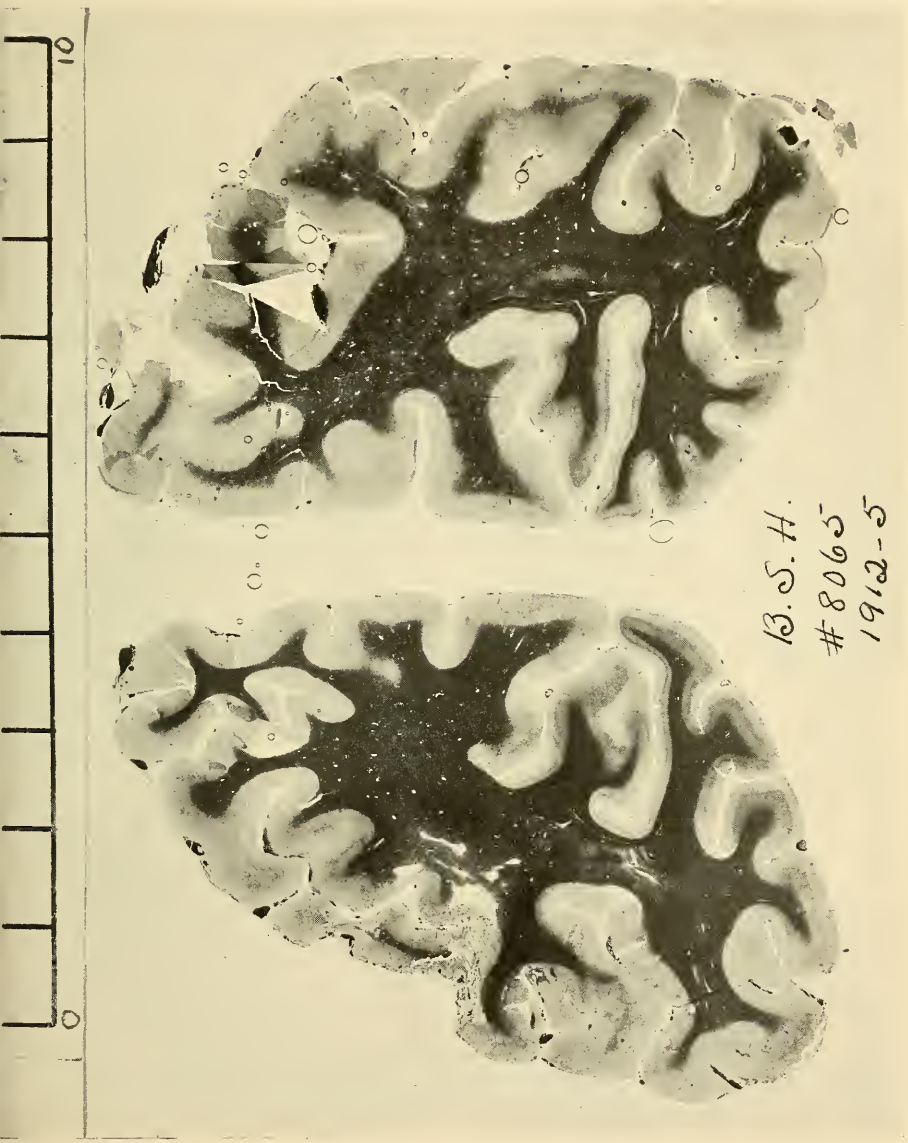

FIG. 1, CASE A.

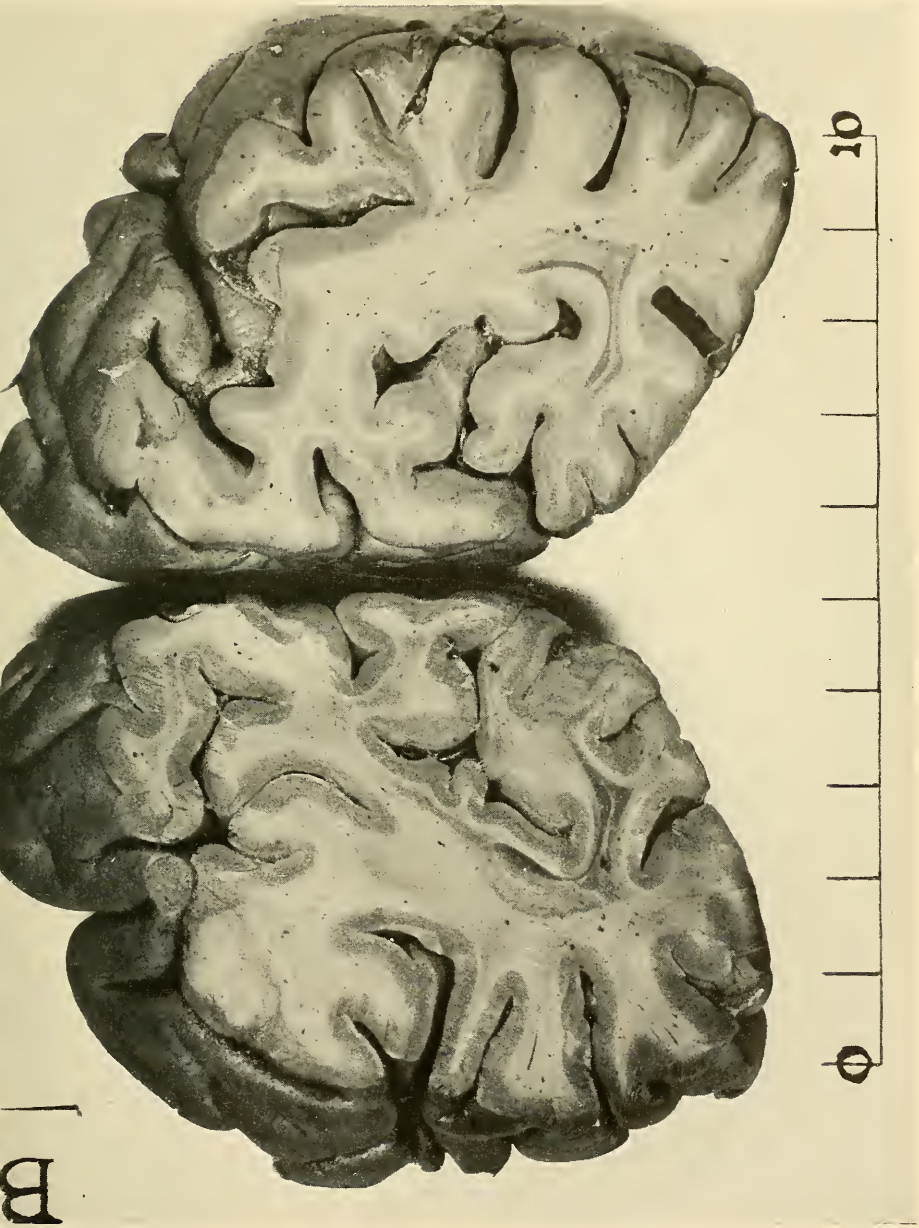

FIG. 2, CASE B.

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A COMPARISON OF THE MENTAL SYMPTOMS FOUND IN CASES OF GENERAL PARESIS WITH AND WITHOUT COARSE BRAIN ATROPHY

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A COMPARISON OF THE MENTAL SYMPTOMS FOUND IN CASES OF GENERAL PARESIS WITH AND WITHOUT COARSE BRAIN ATROPHY[1]

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Most promising leads in psychopathology accrue from the well-known neuropathological desire to prove "structural" as many of the so-called "functional" psychopathies as possible. Though the search for truly functional psychopathies—judged by the hard tests of the post-mortem room—has to be very keen, and though the sure and uncomplicated natural experiments which bring to the post mortem room suitable cases for crucial examination are singularly rare, yet the structuralizing neurologist has not yet come at all near to destroying the functionalist hypothesis. The position that mental disease may well be a disease of function involving no more than normal and inevitable physiological changes in the nervous system is still perfectly tenable, perhaps even correct for some cases. For some time now I have been publishing in various medical journals a number of contributions to the study of normal-looking brains in psychopathic subjects. My associates and I have reported on all available material at various Massachusetts hospitals for the insane (Taunton,[1] Worcester,[2] Westborough,[3] Boston[4]) and have made numerous references[5,6,7] to the largest material (Danvers) which remains as yet unpublished. A large amount of work has had to be done in this search for psychoses that shall be above reproach as to their functionality. As an instance of the intriguing nature of the problem, I may say that out of 153 carefully examined cases at Boston State Hospital, Dr. Canavan and I were able to find but five entirely suited to crucial microscopic examination

[1] Being Contributions of the State Board of Insanity, Number 38 (1915.4). (*Bibliographical Note.*—The previous contribution was S. B. 1. Contributions Number 37 (1915.3) by M. M. Canavan, entitled "A Histological Study of the Optic Nerves in a Random Series of Insane Hospital Cases," Journal of Nervous and Mental Disease, March, 1916.)

and that an orienting examination of these cases with the microscope has already led to disquieting suspicions.[8]

One word is due those who take the advanced and (in my opinion) entirely correct ontological view that structure and function are in such very intimate dyadic relation that they form to all intents and purposes a unity. Such a conception I have tried inadequately to develop in previous communications.[5,6] I trust that the present series of studies will be permitted to rest outside the limits of ontological discussion.

Logically interesting, however, is the progress which can be made by the simple device of cutting an autopsy series or a clinical series in twain on the lines of supposed functionality and structurality. It may be conceded that many cases get pushed to the wrong side of the line, being called structural when they are really (on the present conception) functional, and *vice versa*. But these errors prove themselves in a manner familiar to those employing the statistical method.

The readers of this JOURNAL may recall certain papers on delusions written by Stearns, Tepper, and myself.[9,10,11] In two of these papers the hypothesis was raised that the various (non-paretic) cases in question were really "functional" in the prevailing sense of cases without neural lesions. In a third paper I resorted to material which had to be regarded as "structural," viz., general paresis; but the conclusions founded thereon depend at least as much on the prevailing mode as did my former conclusions on somatic[9] and environmental[10] delusions in "normal-looking brain" cases.

How many of the symptoms of general paresis can safely be correlated with the lesions of general paresis as we know them? This question is exceedingly important, dealing as it does with that mental disease about which perhaps we know the most.[12,13,14] The error in diagnosis is low,[15,16,17] especially if compared with the error in psychiatric diagnosis at large,[18,19] and the number of variables in our equations is correspondingly reduced.

In the study just mentioned[11] we concluded that the *characteristic* delusions of general paresis (found in 57 per cent. of all cases in a routine series, and in 75 per cent. of all cases showing delusions) are delusions about the patient's personality and that these delusions could be roughly correlated with frontal lobe lesions (non-autopsychic delusions failing to be so correlated). These conclusions were in general harmony with findings in dementia præcox.[20,21]

For the present purpose I have split a certain series of autopsied paretic cases in twain on the basis of their showing or not showing substantial gross brain lesions. The series was chosen on the basis of personal examination by me at autopsy and of careful registration of all gross lesions found. The descriptions made were very particular and well-nigh finical, since they were from the beginning destined to be compared with gross findings in various psychoses at one time commonly regarded as functional (dementia præcox, manic-depressive insanity). Without here considering the medically and therapeutically interesting fact that in this random series 18 brains showed no substantial gross lesions and a bare majority, 20, yielded such lesions, I shall proceed to a brief symptom analysis from a psychopathological point of view, reserving for publication elsewhere[22] various medical implications of the work. All cases, both with and without *gross* lesions, possessed the characteristic microscopic lesions developed by the Nissl-Alzheimer school.

Before tabulating the symptoms found in the two "normal-looking" and "abnormal" brain groups or in what might be termed the "mild" and "severe" cases, I must add that we are in no sense dealing with early and late phases of the disease. In fact the mild cases are often the longest cases. There is no question of a progressively severer disease in many cases. The cases progress, it is true, in one sense toward their death, and they do not very often regress. Moreover stationary cases are rarities. But a case lasting five years is not necessarily an anatomically or histologically severer case than one lasting two years.

In explanation of the first two tables, I must premise that (1) The fourth columns contain the number of symptoms (named in the first column) found and catalogued in a series of 17,000 cases clinically analyzed at Danvers State Hospital, only a small portion of which have ever come to autopsy and many of which are still alive. The analysis does not pretend to weigh the importance of the symptoms listed or their dominance in the various cases. The 17,000 list is purely a frequency list. (2) The entries in the *second* column (mild) of Table I represent symptoms in their order of frequency in a series of 18 anatomically "mild" cases of general paresis, whereas in the *third* column (severe) of Table I appear symptoms in their order of frequency in 20 anatomically "severe" cases. (3) The entries in the *second*

column (severe) of Table II represent frequencies in the 20 anatomically "severe" cases and those in the *third* column (mild) the corresponding frequencies in the anatomically "mild" cases.

It occurs to me that some question may well be raised whether anatomical appearances can be safely trusted to gauge severity of processes. Certainly we are aware that in certain cases these appearances can *not* be trusted. But I assume that there can be no doubt that, by and large, the atrophic brain is more deeply affected than the normal-looking brain. At any rate it is a question whether the microscope can be trusted much farther quantitatively at the present time. And in any event the findings both anatomically and symptomatically indicate two groups of cases, whether we choose to regard them as "mild" and "severe" or not.

Without entering the total field of symptomatology in psychiatry, I may perhaps add that I do not necessarily approve the nomenclature of symptoms here adopted and merely record the entries as they stand. The influences of Kraepelin and of Wernicke are plain in the nomenclature, despite the fact that a majority of the facts were collected before the work of either of these masters had come into close contact with practical American psychiatry.

Those symptoms have been included in all columns which occurred in 20 per cent. or more of any of the three series.

Table I

Symptoms Arranged in the Order of those Most Frequent in the Anatomically Mild Cases

	18 Mild	20 Severe	17,000
Amnesia	11	11	*3,422*
Motor restlessness	10	11	*5,428*
Disorientation	10	10	2,419
Delusions, allopsychic	9	3	*6,844*
Dementia	8	9	*5,841*
Depression	7	9	*5,015*
Irritability	7	6	2,714
Defective judgment	7	8	2,596
Psychomotor excitement	6	5	*6,903*
Delusions, autopsychic	6	7	*4,897*
Destructiveness	6	1	2,362
Resistiveness	6	3	2,051
Insomnia	5	4	*4,354*
Violence	5	2	3,244
Aphasia	5	9	1,180
Hallucinations, not specified	5	6	885
Convulsions	5	4	413
Hallucinations, visual	4	6	3,186
Sicchasia	4	2	1,597

I have italicized those figures in the 17,000 columns which represent 20 per cent or more of the 17,000.

Table II

Symptoms Arranged in the Order of those Most Frequent in the Anatomically Severe Cases

	20 Severe	18 Mild	17,000
Amnesia	11	11	*3,422*
Motor restlessness	11	10	*5,428*
Disorientation	10	10	2,419
Dementia	9	8	*5,841*
Depression	9	7	*5,015*
Aphasia	9	5	1,180
Defective judgment	8	7	2,596
Delusions, autopsychic	7	6	*4,897*
Irritability	6	7	2,714
Hallucinations, not specified	6	5	885
Hallucinations, visual	6	4	3,186
Euphoria	6	3	590
Psychomotor excitement	5	6	*6,903*
Incoherence	5	3	*4,130*
Confusion	5	1	2,120
Expansiveness	5	2	386
Insomnia	4	5	*4,354*
Convulsions	4	5	413
Exaltation	4	2	1,711

If we regard the ten statistically leading symptoms in the 17,000 cases as the most frequent of all psychiatric symptoms, and possibly as the most important (although I do not assert the latter), then it is of interest to inquire how far paresis partici-

Table III

Symptoms Arranged in the Order of those Most Frequent in 17,000 Cases

	17,000	18 Mild	20 Severe
Psychomotor excitement	6,903	6	5
Delusions, allopsychic	6,844	9	3
Dementia	5,841	8	9
Hallucinations, auditory	5,428	2	1
Motor restlessness	5,428	10	11
Depression	5,015	7	9
Delusions, autopsychic	4,897	6	7
Insomnia	4,354	5	4
Incoherence	4,130	3	5
Amnesia	3,422	11	11
Violence	3,244	5	2
Hallucinations, visual	3,186	4	6
Irritability	2,714	7	6
Defective judgment	2,596	7	8
Disorientation	2,419	10	10
Destructiveness	2,362	6	1
Confusion	2,120	1	5
Resistiveness	2,051	6	3
Delusions, somatic	1,829	0	0

pates in the nature of mental disease at large and how far it is differentiated on this statistical basis.

The following tables bring out the answer:

In a fourth table I have placed the symptoms in order of frequency as they occurred in 17,000 cases of mental disease analyzed at the Danvers Hospital. The first ten of these symptoms occurred in at least 3,400 cases, that is, in 20 per cent. or more of the series, and the remaining nine are added to secure a statistical parallel to the facts in Tables I and II.

TABLE IV

Mental Disease in General	General Paresis	
	Anatomically Mild	Anatomically Severe
1. Psychomotor excitement	9th to 12th	13th to 16th
2. Allopsychic delusions	4th	Not in first nineteen
3. Dementia	5th	4th to 6th
4. Auditory hallucinations	Not in first nineteen	Not in first nineteen
5. Motor restlessness	2d	2d
6. Depression	6th to 8th	4th to 6th
7. Autopsychic delusions	9th to 12th	8th
8. Insomnia	13th to 17th	17th to 19th
9. Incoherence	Not in first nineteen	13th to 16th
10. Amnesia	1st	1st
11. Violence	13th to 17th	Not in first nineteen
12. Visual hallucinations	18th or 19th	9th to 12th
13. Irritability	6th to 8th	9th to 12th
14. Defective judgment	6th to 8th	7th
15. Disorientation	3d	3d
16. Destructiveness	9th to 12th	Not in first nineteen
17. Confusion	Not in first nineteen	13th to 16th
18. Resistiveness	9th to 12th	Not in first nineteen
19. Somatic delusions	Not in first nineteen	Not in first nineteen

Analysis of this table shows that *auditory hallucinations* and *somatic delusions* are the only symptoms which, while appearing amongst the first nineteen symptoms of mental disease in general, fail to appear among the first nineteen symptoms of general paresis in either the mild or the severe group. It will be remembered that the first nineteen symptoms in general paresis were chosen as occurring in at least 20 per cent. of the cases studied, and that but ten symptoms in mental disease at large occur in over 20 per cent. of cases. Hence the failure of *auditory hallucinations* to occur in any considerable number of cases of paresis is made more striking than the absence of *somatic delusions*. The presence of *visual hallucinations*, to be sure at the bottom of the list among mild cases, but in fair proportion among severe cases, is theoretically hard to explain, when taken in conjunction with the paucity of *auditory hallucinations*. Indications in the literature point perhaps to optic nerve lesions as

a possible basis for the *visual hallucinations,* suggesting an almost illusory origin therefor.

The fact that *allopsychic delusions* are so common, at least in the mild cases, seems to show that they are not correlated with *auditory hallucinations* either as cause or effect. It is as if there were not even pseudoreality to the *allopsychic delusions* and as if they did not appear even to the patient as representing centripetal (*e. g.,* hostile) effects. In fact, as will appear below, these *allopsychic delusions* are associated more with refusal of food (hallucinatory tastes ?, comments on indigestion?) than with *auditory hallucinations.* The study of *allopsychic delusions* in the paretic ought therefore to present conceptions of a quite disparate order to those of the victim of dementia præcox, where *auditory hallucinations* are so characteristic (see recent redeterminations of a statistical nature by Stearns[23]).

The paucity of *somatic delusions* in both paretic groups is perhaps not surprising and is in line with some previous determinations including those of Southard and Tepper.[11] The peripheral origin of many somatic delusions or at all events their strong peripheral element, as claimed in previous papers,[9,24] is consistent with this determination. The presence of a fair proportion of *visual hallucinations* remains astounding except on the basis of optic nerve changes mentioned above. Since Canavan[25] has shown a high proportion of chronic optic nerve changes in routine autopsied cases of all sorts of mental disease (paretic and non-paretic), it might be argued that *visual hallucinations* should be more common in mental disease at large. In point of fact *visual hallucinations* do seem to stand somewhat higher in order of frequency in mental disease at large than might have been *a priori* supposed. But, why, if *visual hallucinations* are really related (as some assert) with peripheral nerve changes, should not *tactile* and other *haptic hallucinations* occur more frequently in general paresis, in which the peripheral nerves are not infrequently involved? Perhaps such *haptic hallucinations* do occur but fail to reach the medical observer.

The agreement of both paretic groups in placing *amnesia, motor restlessness,* and *disorientation* in one, two, three order is of great interest. If we omit the anomalous *allopsychic delusions* from the mild group for the moment, then *dementia* would follow as a fourth common symptom. Further discussion is placed below.

For the purposes of Table IV we extended the list of symptoms from mental disease at large to nineteen for comparison with the nineteen symptoms which we had found to occur in over 20 per cent. of all cases of paresis. As a matter of fact the two lists of nineteen symptoms in paresis are not identical, and the differences are instructive.

The following are symptoms which occur in over 20 per cent. of the mild cases that do not occur in 20 per cent. of the severe cases.

Allopsychic delusions	9 in 18	3 in 20	6,844 in 17,000
Sicchasia	4 in 18	2 in 20	1,597 in 17,000
Resistiveness	6 in 18	3 in 20	2,051 in 17,000
Destructiveness	6 in 18	1 in 20	2,362 in 17,000
Violence	5 in 18	2 in 20	3,244 in 17,000

I have arranged the list arbitrarily on the basis of a vague conception of the interrelation and possibly the intergrading of some of these symptoms. I believe their mutual relations are plain: *the mild case of paresis,* in more than a fifth of all cases and often in far more than a fifth, *is reacting to his environment* (especially to his personal entourage) *most markedly.* Let us glance at the symptoms which distinguish the anatomically severe from the mild cases, since they fail to occur in 20 per cent. of the latter.

Euphoria	6 in 20	3 in 18	590 in 17,000
Expansiveness	5 in 20	2 in 18	386 in 17,000
Exaltation	4 in 20	2 in 18	2,711 in 17,000
Confusion	5 in 20	1 in 18	2,120 in 17,000
Incoherence	5 in 20	3 in 18	4,130 in 17,000

Here again, just as perhaps we might separate two symptoms (*allopsychic delusions* and *sicchasia*) from the other three which form a group by themselves among the distinguishing features of the "mild" group, so we may separate *confusion* and *incoherence* from the other three mutually related symptoms, *euphoria, expansiveness,* and *exaltation* in the "severe" group.

It was the observation of this contrast which caused me to write out the present paper for this JOURNAL, since I felt there was a general psychopathological interest to the contrast, which must very probably be based on structural differences in disease-process.

I have throughout left the impression that the structural differences in the two groups are largely those of extent. Perhaps

extent, depth, and serial involvement of cortex layers may indeed have something to do with these functional differences. Histological studies of striking instances of these phenomena may well confirm one or other of these conceptions.

Meantime we should also take into account the habitual preference of gross brain lesions in general paresis for the frontal region. With this fact in mind, a somewhat speculative account of the situation might run to this effect: That the *severe cases* with gross brain involvement *tend to leave the parietal regions* relatively intact and subject to operations *unchecked by the great inhibitory frontal areas.* The expansiveness of the paretic would accordingly resemble the hyperphantasia of certain victims of dementia præcox. The latter I have been trying to associate with the mild atrophic lesions of the parietal regions which affect certain cases of dementia præcox.[21] General paresis very probably often possesses similarly mild lesions of the parietal regions, differing from those of dementia præcox in being exudative rather than merely degenerative. But at a time when these parietal lesions are beginning to develop in paresis, the frontal regions are doubtless often far on the road to coarse atrophy. Inhibitory power the frontal regions no longer possess, certainly over many motor activities, possibly over various conceptual processes. Thus might be explained both the resemblances and the divergences of hyperphantasia (fantastic delusions) and expansiveness (delusions of grandeur).

But now, as has been stated, a large minority of cases of paresis fail to die with coarse brain atrophy. All these cases have exudative lesions of more or less prominence, despite the absence of coarse brain atrophy. Just as the mild lesions of the parietal regions may produce (virtually as irritative symptoms) *expansiveness* and attendant *euphoria* and *exaltation* at the same time as coarse frontal destruction is leading to *confusion, incoherence,* and a disintegration of the patient's entire attitude to men and things, so the mild lesions of the frontal region may be leading to the above mentioned *anti-environmental* group of symptoms in the non-atrophic group. Action is not inhibited in its entirety or in its coarser manifestations. The operation of an exudative (and not yet extremely destructive) lesion in this frontal area may act in part to abolish the inhibitions which are very possibly the proper function of this area, but may also act in part to irritate, interrupt, and throw into disorder those inhibitions. The mild microscopic lesions in these non-atrophic cases

may act to bring about not the classical loss of inhibition but a perversion of inhibition, an incoördinate and irregular checking of activities, and of those *in*activities which proper conduct often requires. On such lines could be explained with some plausibility the *resistiveness, destructiveness,* and *violence* which appear to be characteristic of these non-atrophic cases.

As to an explanation of the *delusions of persecution* and *refusal of food,* the situation is perhaps not so clear. The *sicchasia* may sometimes be an example of *resistiveness* and again due to delusions. If the former, then the symptom would best be explained as the result of disorder of inhibition. If the latter, I can only offer the analogy of dementia præcox, in which for some reason or other delusions (except fantastic) are rather closely associated with frontal lobe lesions. The psychopathology of delusions is obscure. I hold the opinion, however, that delusions represent more a disorder of believing than a group of false beliefs, rather more a perversion of volitional process than of intellectual process. On this line of reasoning I find it somewhat easy to reconcile the relation of the mild frontal lesions here found to delusions about the environment. Thus I would align together all five of the distinctive symptoms of the mild group with perversions of inhibition, presumably largely due to frontal lobe lesions even though these are hardly or not at all represented in the gross. In cases with more extensive frontal lobe destruction (coupled often perhaps with the establishment of *mild* lesions elsewhere in the cortex), the perversions of inhibition are replaced by frank losses thereof: the anti-environmental tendencies of the mild cases are replaced by less socially disturbing yet more profound disorder of personality.

Summary and Conclusions

The possession of a suitable statistical background (The Danvers Case Symptom Index) has rendered worth while an orienting study in the mental symptomatology of general paresis. A group of 38 general paretics whose brains were specially examined and described by the writer, has been divided into two groups according to whether there was or was not coarse evidence of brain atrophy. The cases without brain atrophy were termed "mild" and those with brain atrophy were termed "severe," although these designations are only approximations to accuracy; the groups are, however in no sense "early" and "prolonged."

Symptomatically the two groups show several surprising concordances and a number of instructive divergences. Thus *amnesia, motor restlessness, disorientation, dementia,* and *depression* lead both series and in that order (except that *allopsychic delusions* stand fourth in the "mild" series and are far less common in the "severe"). *Are amnesia and dementia therefore in no sense proportional to brain tissue loss?*

Nineteen symptoms occurred in 20 per cent. or over of the paretic series, viz., the five just mentioned, and *nine* others (*irritability, defective judgment, psychomotor excitement, autopsychic delusions, insomnia, aphasia, hallucinations* of doubtful or unspecified nature, *convulsions, visual hallucinations*) not always in like proportion in the two series. *Five* other symptoms occurred in each series, but symptoms quite sundered from one another in general significance.

The "mild" cases showed a group of symptoms which might be termed *contra-environmental,* viz., *allopsychic delusions, sichasia* (refusal of food), *resistiveness, violence, destructiveness,*

The "severe" cases showed a group of symptoms of a quite different order, affecting *personality,* either to a ruin of its mechanisms in *confusion* and *incoherence,* or to the mental quietus involved in *euphoria, exaltation,* or *expansiveness.*

Some speculations are offered in the text as to the perversion of inhibition or incoördination of inhibition which the largely irritative lesions of the "mild" cases are presumably effecting in the perhaps more seriously affected frontal areas. When these are still more gravely affected, as to the point of atrophy, then the intrapsychic disorder might well become more manifest, *e. g.,* in the distinctive symptoms of the "severe" group just mentioned.

In a series of 17,000 clinical cases (of all sorts of mental disease, alive and dead, recovered and impaired) symptomatologically analyzed, there were but *ten* symptoms occurring in 20 per cent. or over; These were in order, *psychomotor excitement, allopsychic delusions, dementia, auditory hallucinations, motor restlessness, depression, autopsychic delusions, insomnia, incoherence, amnesia.* Each of these is represented high in general paresis (*i. e.,* in 20 per cent. or over) except that *auditory hallucinations* are infrequent in both "mild" and "severe" cases and *allopsychic delusions* are infrequent in "severe" cases. There may be topographical reasons for the paucity of *auditory hallucinations* in general paresis. The method of production of

allopsychic delusions in general paresis should be studied, since there can be no such alliance of *allopsychic delusions* and *auditory hallucinations* therein as is perhaps the rule in dementia præcox.

If we consider the next *nine* symptoms in order in 17,000 cases of mental disease at large, viz., *violence, visual hallucinations, irritability, defective judgment, disorientation, destructiveness, confusion, resistiveness,* and *somatic delusions,* we find only the last, viz., *somatic delusions,* not represented in either group in fair proportion, although (as above stated) *confusion* is poorly represented in the "mild" cases and *violence, destructiveness,* and *resistiveness* are poorly represented in the "severe" cases.

Aphasia, hallucinations of doubtful or unspecified nature, and *convulsions* appear to be frequent symptoms in general paresis that do not figure at all so largely in mental disease as a whole. Besides these, *sicchasia* of the "mild" group and *euphoria, exaltation,* and *expansiveness* of the "severe" group appear to stand out for general paresis against mental disease as a whole.

The most positive results of this orienting study appear to be the unlikelihood of *euphoria* and allied symptoms in the "mild" or non-atrophic cases and the unlikelihood of certain symptoms, here termed *contra-environmental,* in the "severe" or atrophic cases. Perhaps these statistical facts may lay a foundation for a study of the pathogenesis of these symptoms. Meantime the pathogenesis of such symptoms as *amnesia* and *dementia* cannot be said to be nearer a structural resolution, as these symptoms appear to be approximately as common in the "mild" as in the "severe" groups.

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LII

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ON THE APPLICATION OF GRAMMATICAL CATEGORIES TO THE ANALYSIS OF DELUSIONS.

ABSTRACT.

Remarks on Royce's sociological and logical influences. The general nature of Royce's logical seminary; choice of topics. As to the superposition of grammatical upon psychiatric concepts, the reason for choosing delusions. Delusions in the Danvers symptom catalogue and their place in nosological entities. The neglect of delusions by logic and psychology. James's handling of delusions probably over-sensationalistic. Probable value of the psychopathological point of view as illustrated in James's later work. Analysis of certain instances of somatic delusion. Analysis of certain instances of environmental and personal delusions. Contrasting results of the somatic and personal group analyses. Anatomical intimations that the frontal lobes are involved more especially in disorder of personality. Function of impression more likely to employ posterior-lying nerve tissue; function of expression, anterior-lying. Two groups of delusions in dementia præcox, one associated with frontal lobe anomalies or lesions, the other with parietal: the latter delusions fantastic. The pragmatic element in most delusions invites comparison with the grammatical categories of the verbs. Delbrück *vs.* Wundt *re* grammar and psychology. Non-identity of these topics. The four fundamental moods (imperative, indicative, subjunctive, optative). Subjunctive the mood of *will*, optative that of *wish*. 'Stratified' development of these moods. Their relation to human character types. Relation of grammatical moods to logical modality (necessary, impossible, contingent, possible). Importance of getting a clear conception of beliefs from the point of view of the believer. Category of the voice (active, passive, middle). Situation passive with many hallucinations, perhaps reflexive in the case of *Gedankenlautwerden*. Involvement of the first person. Importance of distinguishing the second from the third person from the patient's point of view. Gender and number of persons involved in a delusional situation. Do essentially tetradic situations occur, at least where the number of persons involved is *manifestly* four? Tense-distinctions. Probability that most moods with special names in different languages fall toward either the subjunctive (*e. g.*, potential, conditional) or the optative (*e. g.*, desiderative, precative, jussive?). Pragmatic delusions as subjunctive 'precipitates.' Fantastic delusions as optative 'precipitates.' Summary.

I.

I AM peculiarly glad to speak here in honor of Royce. Especially in recent years I have felt, in my professional work as neuropathologist and as psychiatrist, the effects of Royce's teaching, more particularly of his graduate teaching in the logical seminary, which I have followed omitting a few years only since

1897. I well remember when my training with James and Royce was regarded as something of a disability: it was questioned whether a man with philosophical antecedents could do the work of an interne in pathology! Nowadays we have pretty well worked through that period to one of greater tolerance.

I want to illustrate in this paper a concrete effect of Royce's logical seminary through the employment of its comparative method in a certain special field of psychiatry wherein are to be applied some categories derived from a portion of the science of grammar.

But first a word as to broader effects of Royce's work. I do not speak of his metaphysics, except as it has relation to the social consciousness. My colleague, Richard Cabot, has already to-day spoken of the Royce influence upon himself. In more limited ways, I must own to identical influences, making for a greater interest in social service than is common among physicians. And indeed the sociological influences of Royce have been wide, as may be seen in the chapter "Of Society"[1] in the fourth volume of Merz's *A History of European Thought in the Nineteenth Century*, 1914. Therein Merz sets forth how "no subject of philosophical or scientific interest has been more profoundly affected by it [the spirit of comprehension in opposition to that of definition, or as later termed, the 'synoptic' tendency] than the study of man in his individual and collective existence." After then speaking of new definitions of the social 'Together,' of the 'social self' as opposed to the subjective, Merz ascribes to Royce "the clearest indication of this doctrine," quoting a passage from the papers of Royce contained in early volumes, 1894–1895, of this REVIEW.[2] I have no specialist's command of the history of these developments, but I am sure that the history of Richard Cabot's justly famous campaign for social service could not be written without reference to Royce's work on the social consciousness. And I know personally that hardly a day passes at the Psychopathic

[1] Merz, J. T., *A History of European Thought in the Nineteenth Century*, Vol. IV, Chap. X, "Of Society," p. 437. Blackwood, Edinburgh, 1904.

[2] Royce, J., "The External World and the Social Consciousness." *Philos. Review*, 3, 1894; and "Self Consciousness, Social Consciousness and Nature," *ibid.*, 4, 1895.

Hospital in Boston without concrete exemplification of these interests as opposed to the purely medical.[1]

What I wish here to set forth is a matter of special psychiatric analysis whose scope and shape have been transformed by influences, not so much of a sociological, as of a logical nature, drawn from Royce's seminary. That seminary has dealt with a great variety of topics from a comparative point of view, although the statistical sciences have not been neglected. Such widely contrasting points of view as those of L. J. Henderson (revolving about the considerations of his book on *The Fitness of the Environment*[2]) and those of F. A. Woods (revolving about the considerations of his books on *The Influence of Monarchs*[3] and *Is War Diminishing?*[4]) have been brought by their authors in the developmental state to the seminary.

The topics of the Seminary over a long period of years have been well-nigh as wide in range as those of, *e. g.*, Wundt's *Logik*,[5] but their choice has not been governed by any principle such as that of Wundt's *Logik* or by any evident principle except that of the needs of a variety of workers who have for a variety of reasons been attracted to the Seminary. Accordingly, although the principle of a book like Wundt's majestic volumes on *Logik* is probably to some extent aprioristic, or at any rate governed by still more general metaphysical principles than those which the book itself sets forth, the topics of Professor Royce's Seminary have subjected themselves to no special principle; and this despite the fact that the seminary visitors and its moderator have often been tempted into metaphysical digressions. Aside from the personality of the leader, very possibly the effects of the thought of the late Charles S. Peirce and the late Professor

[1] (Southard, E. E., editor), *Contributions from the Psychopathic Hospital* (Department of the Boston State Hospital), Boston, Mass., 1913 and 1914.

[2] Henderson, L. J., *The Fitness of the Environment, an Inquiry into the Biological Significance of the Properties of Matter*, Macmillan, N. Y., 1913.

[3] Woods, F. A., *The Influence of Monarchs, Steps in a New Science of History*, Macmillan, N. Y., 1913.

[4] Woods, F. A., and Baltzly, A., *Is War Diminishing? A Study of the Prevalence of War in Europe from 1450 to the Present Day*, Houghton, Mifflin, Boston, 1915.

[5] Wundt, W., *Logik, Eine Untersuchung der Principien der Erkenntnis unter der Methoden wissenschaftlicher Forschung*, 3 Aufl., Stuttgart, Enke, 1903.

William James have been most in evidence; more particularly, perhaps, the effects of Peirce's thought.

II.

My special topic may be described as a *grammar of delusions*, or more exactly as an application of a portion of the logical classifications of grammar (and more especially the grammar of verbs) to a portion of the data of psychiatry, viz., delusions (and more especially certain delusions that I call pragmatic or parapragmatic to distinguish them from fantastic or more purely ideational delusions). The connotation of the term *grammar* is therefore not that of the elementary-and-therefore-simple-and-reliable, which the term receives in, say, Newman's *Grammar of Assent* or Pearson's *Grammar of Science.*

My reason for choosing delusions as one member of the comparative system which I proposed to employ as illustrative of the method of Royce's seminary was as follows. First, there was no doubt from an inspection of the records of state hospitals for the insane that delusions or false beliefs of many sorts were among the most frequent of psychopathic phenomena. Secondly, it did not appear that the topic had been taken up seriously either by logic or by psychology.

First, to develop a little farther the frequency of delusions amongst the insane, I may refer to the data of the Danvers (Massachusetts) State Hospital symptom catalogue, unique I believe in its representativeness of routine records of comparatively high standard.[1] Despite the fact that many patients do not exhibit definite delusions of a nature permitting accurate transcription, yet in some 17,000 cases of all sorts of mental disease examined at the Danvers State Hospital, period of 1879 to 1913,[2] there were certainly no less than 5,000 cases in which the delusions were definite enough to permit being recorded in the case history. No doubt this experience is the pre-

[1] Southard, E. E., *The Laboratory Work of the Danvers State Hospital, Hathorne, Massachusetts. With especial Relation to the Policy Formulated by Dr. Charles Whitney Page, Superintendent, 1888–1898, 1903–1910.* Boston Med. Surg. Jour., vol. clxiii, pp. 150–227, Aug. 4, 1910.

[2] Southard, E. E., *A Study of Normal-looking Brains in Psychopathic Subjects, with Notes on Symptomatology* (Danvers State Hospital Material) to be published.

vailing one, and no doubt more intensive histories would greatly augment the percentage of cases characterized at one time or other by delusions.

Such figures of course far transcend the numbers of true 'paranoiacs' (or even victims of paranoid forms of the dementia præcox of Kraepelin), and I should not wish to be understood to say that, in the 5,000 or more Danvers cases, delusions formed the head and center of the mental diseases in question.

Yet the number of actual entities (in the medical sense of this term as a kind of collection of symptoms) in which delusions do form a central feature makes a formidable list. I may limit myself to the following actual or possible entities: paranoia, the paranoid form of dementia præcox, and the somewhat closely allied paraphrenia of Kraepelin's recent formulation, the so-called acute alcoholic hallucinosis, or insanity of alcoholic origin, a number of forms of pre-senile psychoses, some forms of senile psychoses, to say nothing of various forms of syphilitic mental disease, as also manic depressive psychosis, various mild or severe psychopathic conditions not ordinarily considered to amount to frank mental disease, and even such apparently remote entities, or groups of entities, as are found under the caption of epilepsy and feeblemindedness.

So much will suffice to show the frequency of delusions among psychopaths and the probable magnitude of the problem for the science of psychiatry. I need not here discuss the somewhat large psychiatric literature of delusions. I confess that the literature in question has struck me as a little barren or at best the threshing over of old straw by the application of categories borrowed, *e. g.*, from Herbart or Wundt to material that neither had ever concretely considered.

Secondly, to develop a little farther the logical and psychological neglect of the topic. The logic of fallacies, *e. g.*, in Alfred Sidgwick's excellent work,[1] makes not the slightest draught upon psychiatric data, not merely perhaps because the delusions of

[1] Sidgwick, A., *Fallacies, a View of Logic from the Practical Side*, The International Scientific Series, Appleton, N. Y., 1884. *Distinction and the Criticism of Beliefs*, Longmans, Green, London, 1892.

the insane are not prominently fallacious (at least some of the most serious and important of insane delusions) but because a logician would never spontaneously think of going to psychiatry for logical material.

But also and more markedly perhaps, it would be somewhat easy to show that delusions, especially of the insane, have been too largely neglected by the psychologists. Even James, in whose work may be seen remarkable influences of his psychopathological point of view, deals with delusions of the insane in a very few brief pages.[1] For example, he cites insane delusions along with alternating selves and mediumships as a type of abnormal alterations in the self, quoting Ribot upon our personality and Griesinger upon the 'doubleness' of self, of the 'struggle of the old self against new discordant forms of experience,' 'the opposition of the conscious me's,' etc. Again, James quotes from Krishaber a case of the well-known metaphysical type of delusions with feelings of unreality. In a footnote to his chapter on the perception of things, James quotes a list of certain special delusions given by Clouston, suggesting that in many cases "there are certain theories which the patients invent to account for their abnormal bodily sensations," "that in other cases they are due to hallucinations of hearing and sight." James here also defines a delusion "as a false opinion about a matter of fact which need not necessarily involve, though it often does involve, false perceptions of sensible things."

How rationalistic, nay sensationalistic, are these latter definitions just quoted from James! The point is urged that the data of reasoning are as it were poisoned at the sensory source. Theories are invented, or hallucinations supply data.

This, as it seems to me, over-rationalistic account of delusions is the more remarkable in James because the whole trend of his thinking was surely bent by his medical or psychopathological point of view. Those of us who have confidence in the psychopathological method may indeed feel that the key to a thoroughgoing theory of belief may be found in a study of delusions; namely, of false beliefs.

[1] James, W., *The Principles of Psychology*, Henry Holt, N. Y., 1890, Vol. II, Chap. XIX, "The Perception of 'Things,'" footnote, p. 114.

I should like to dwell on the James point of view here, because I think his progress subsequent to the *Principles of Psychology* and culminating in *The Varieties of Religious Experience*[1] shows a drawing-away from the sensationalistic point of view to a very overt voluntarism, under which, had James considered the problem of delusions, he might well have dealt with them as perversions of will rather than false conceptions or conceptions based on false perceptions, hallucinations, or strange bodily sensations.

It is difficult not to think that the logical method at the bottom of James's *Varieties of Religious Experience* is not essentially the method of psychopathology despite the careful guarding of the point of view from certain misconstructions in the initial chapter of that work, entitled "Religion and Neurology." As when James states concerning the phenomena of religious experience that "When I handle them biologically and psychologically as if they were mere curious facts of individual history, some of you may think it a degradation to so sublime a subject and may even suspect me, until my purpose gets more fully expressed, of deliberately seeking to discredit the religious side of life." James, it will be remembered, furnishes a concrete example in George Fox, pointing out that whereas the Quaker religion, which he founded, is something which it is impossible to overpraise, yet Fox's mind was unsound, and from the point of view of his nervous constitution, he was a psychopath or "detraqué of the deepest dye."

To be sure, we do not need to guard the results of an analysis of insane delusions with such cautious remarks as the above concerning the psychopathic varieties of religious experience. Yet I am inclined to believe that whether or no the point of view of psychopathology is more important than that of the classical psychology in the analysis of belief, at any rate the possible contributions of psychopathology have been singularly neglected.

Accordingly, some years ago I started some superficial and

[1] James, W., *The Varieties of Religious Experience, A Study in Human Nature.* Being the Gifford Lectures on Natural Religion delivered at Edinburgh in 1901–1902. Longmans, Green, London, 1902.

orienting analyses of delusional material,[1] the results of which I wish to present briefly here, partly to show the general nature of the material.

My first systematic work dealt with somatic delusions[2] and the result was decidedly sensationalistic and quite aptly illustrated James's remark above quoted concerning "theories which the patients invent to account for their abnormal bodily sensations." In fact it was only when one passed from somatic to personal and environmental delusions that what I have called the sensationalistic hypotheses seemed to fail.

To quote a portion of the conclusions drawn from the work on somatic delusions, "the concept of the *crystallization of delusions around sensorial data of an abnormal sort* must be entertained for some delusions at least." More in detail, "In one group of cases (Cases I, II, III, possibly VIII) the psychic rendering of the somatic states is rather critical and temporary, and follows a process somewhat comprehensible to the normal mind. (Type: "*shot by a fellow with a seven-shooter*," in a *spot found to correspond with a patch of dry pleurisy.*)"

"In others (Cases IV, V) the psychic rendering is less natural and is more a genuine transformation of the sensorial data into ideas quite new. (Type: "*bees in the skull*" found in the case with cranial osteomalacia.)"

"In others (Cases VI, VII) the problem is raised whether severe hypochondria, with ideas concerning dead entrails and the like, may not often indicate such severe somatic disease as tuberculosis. The psychic rendering here is of a more general (apperceptive?) sort."

A somewhat generalized account of this conception was presented in more popular form by my friend Dr. Franz in the *Popular Science Monthly*.[3]

[1] Southard, E. E., and Mitchell, H. W., "Melancholia with Delusions of Negation: Three Cases with Autopsy," *Jour. Nervous and Mental Disease*, 1908, Vol. 35.

Southard, E. E., and Fitzgerald, J. G., "Discussion of Psychic and Somatic Factors in a Case of Acute Delirium Dying of Septicemia," *Boston Medical & Surgical Journal*, 1910, Vol. 162.

[2] Southard, E. E., "On the Somatic Sources of Somatic Delusions." *Jour. Abnormal Psychology*, December, 1912–Jan., 1913.

[3] Franz, S. I., "Delusions," *Popular Science Monthly*, January, 1915.

A second paper on environmental (or, as I called them following Wernicke, allopsychic) delusions[1] yielded the in one sense negative result that environmental delusions seemed to trace back in most instances to temporally or logically prior disorder of personality. I raised then the question whether delusions often spread inwards (egocentripetally) or habitually outwards (egocentrifugally), a concept later to be illuminated by the concept of the voice (active, passive, or reflexive) in which the patients habitually or characteristically moved.

I found that, to quote a later paper on delusions of personality,[2] "put briefly, the deluded patient is more apt to divine correctly the diseases of his body than his devilments by society." Or more in detail "these delusions having a social content pointed far more often inwards at the personality of the patient than outwards at the conditions of the world. And case after case, having apparently an almost pure display of environmental delusions, turned out to possess most obvious defects of intellect or of temperament which would forbid their owners to react properly to the most favorable of environments. Hence, we believe, it may be generally stated that the clinician is far less likely to get valuable points as to the social exteriors of his patients from the contents of their social delusions than he proved to be able to get when reasoning from somatic delusions to somatic interiors."

A word is perhaps necessary to guard against too sweeping conclusions. "In a few cases it seemed that something like a close correlation did exist between such allopsychic delusions and the conditions which had surrounded the patient—the delusory fears of insane merchants ran on commercial ruin, and certain women dealt in their delusions largely with domestic *debâcles*. But, on the whole, we could *not* say that, as the somatic delusions seemed to grow out of and somewhat fairly represent the conditions of the soma, so the environmental delusions would appear to grow out of or fairly represent the environment."

[1] Southard, E. E., and Stearns, H. W., "How Far is the Environment Responsible for Delusions?" *Jour. Abnormal Psychology*, June–July, 1913.

[2] Southard, E. E., "Data Concerning Delusions of Personality. With Note on the Association of Bright's Disease and Unpleasant Delusions," *Jour. Abnormal Psychology*, Oct.–Nov., 1915.

I need quote from only one more paper on the delusion question. The papers above mentioned deal chiefly with cases whose brains looked normal to the naked eye, the material having been chosen as nearest to normal. In another study I deliberately took up perhaps the most abnormal material that we possess in psychiatry, namely, subjects of general paresis,[1] a disease now regarded as a form of brain syphilis. Incidentally I found that the somatic delusions, despite the grave brain damage of paresis, tended to show somatic sources, precisely as had the normal-brain material. When it came to allopsychic (environmental) and autopsychic (personal) delusions, it appeared that these delusions were statistically associated with lesions of the frontal lobes, and that cases without frontal emphasis of lesions were not at all apt to be delusional or, for that matter, to be specially subject to grave disorder of personality.

Now it might not be at once obvious to those who have not followed the progress of brain physiology whither these frontal lobe findings would speculatively lead. I shall develop the matter merely to the point of justifying the choice of the grammar of verbs rather than that of nouns for comparative purposes (I bear in mind that I have not yet justified the choice of grammar at all for such purposes).

There has been, ever since the discovery attributed to Charles Bell of the different functions of the posterior and anterior spinal nerve roots, a growing mass of data concerning the posterior situation of the sensory arrival-platforms (a term of F. W. Mott) and the anterior situation of the motor departure-platforms. The evolutionary complications of the bulb and indeed of the whole rhombencephalon and of the isthmus cerebri did not succeed in abolishing this general tendency to the posterior situation of the sensory arrangements, despite their sidewise pushing in certain regions.

The posterior-lying cerebellum is regarded as a sensory organ despite its indirect chief function of modifying muscular activity

[1] Southard, E. E., and Tepper, A. S., "The Possible Correlation between Delusions and Cortex Lesions in General Paresis," *Jour. Abnormal Psychology*, Oct.–Nov., 1913.

in certain ways. Then the physiologists found a variety of sensory spheres more posteriorly lying in the cerebrum. Sherrington found that the fissure of Rolando had tissue behind it that must be regarded as receptive in nature and tissue forward of it that must be regarded as motor. Moreover, different parts of the precentral gyrus serving face, arm, and leg were found to lie immediately adjacent to receptive tissues for the self-same structures lying back of the Rolandic fissure in the postcentral gyrus.

Accordingly it appeared that the nerve tissues exhibit a somewhat general law to the effect that the function of impression is likely to employ posterior-lying tissues, whereas, anterior-lying tissues are likely to be related with the function of expression, and this law is likely to find expression not alone in the simple spinal cord but also in the complicated cerebral cortex.

It it were permissible to draw psychological conclusions from this law as applied to the cerebral cortex, it might be plausibly mentioned that consciousness, in so far as it is cognitive, whether those cognitions are visual, auditory, or kinæsthetic, is rather more likely to employ posterior-lying tissues than anterior-lying ones in the cortex. Campbell[1] indeed gave utterance to the suspicion that consciousness is a function of the posterior association center of Flechsig. I am personally inclined to this view.

It is clear then that to find delusions related to frontal lobe disorder, *i. e.*, to disorder of forward-lying tissues was at first surprising. Delusions or false beliefs have the ring of consciousness, of cognition, of ideas. The falsity of these ideas is somehow taken as residing in the ideas; at least that is the tendency of the analyst. Hence, if one were seeking cortical correlations for false beliefs taken as ideas essentially and intrinsically false, one would be apt to turn forthwith, not to the *frontal* lobes, but say to the parietal lobes.

Surprises in the nature of results diametrically opposed to expectation are somewhat frequent in neurology as elsewhere. I had been astonished to find, in the obscure quasi-functional but probably in some sense 'organic' disease dementia præcox, that

[1] Campbell, A. W., *Histological Studies on the Localization of Cerebral Function*, Univ. Press, Cambridge, Eng., 1905, esp. p. 206.

the symptom katatonia, a highly motor-looking symptom, tended to associate itself with posterior-lying tissues.[1] In the same disease, delusions tended to relate themselves with frontal lobe lesions. Not only were delusions found to be based as a rule on frontal disease and katatonic symptoms on parietal lobe disease, but an equally strong correlation was found between auditory hallucinations and disease of the temporal lobe. Of course the correlation between auditory hallucinations and lesions of the temporal lobes might be *à priori* expected, but the writer at least did not suspect beforehand the possibility of any relation between katatonia (a condition in which hypertensive states of the muscles occur, sometimes amounting to actual *flexibilitas cerea* and catalepsy) and disease of the parietal region. In point of fact, the strikingly cataleptic cases of my series seemed to be often associated with gross lesions of the post-central gyrus, thus giving rise to a suspicion that the condition katatonia or catalepsy is actually due to a disorder of kinæsthesia, or at all events of the tissues which are in some sense the seat of kinæsthesia. This, then, is an example of one of the perennial surprises of observation. An apparent disorder of motion seems to resolve itself into an actual disorder on the afferent side.

Equally surprising in an opposite direction was the correlation of delusion formation with disease of the frontal lobes. As elsewhere stated in this paper, a rationalistic or sensationalistic account of delusions would naturally lead us to think of brain disorder in the sensorium. In point of fact, the parts of the brain which are best entitled to the name sensorium seem to be free of gross lesions and anomaly except in a comparatively small hyperphantasia group. To quote from conclusions of a paper on Dementia Præcox, "The non-frontal group of delusion-formations the writer wishes to group provisionally under the term *hyperphantasia*, emphasizing the overimagination or perverted imagination of these cases, the frequent lack of any appropriate

[1] Southard, E. E., "A Study of the Dementia Praecox Group in the Light of Certain Cases Showing Anomalies or Scleroses in Particular Brain-Regions," *Am. Jour. Insanity*, Vol. LXVII, 1910–11. On the Topographical Distribution of Cortex Lesions and Anomalies in Dementia Praecox, with some account of their Functional Significance, *Am. Jour. Insanity*, Vol. LXXI, 1914–15.

conduct-disorder in the patients harboring such delusions, and the *à priori* likelihood that these cases should turn out to have posterior-association-center disease rather than disease of the anterior association-center. This anatomical correlation is in fact the one observed."

To sum up the argument to this point, delusions of the insane have been chosen for comparative study because of their frequency as symptoms and their centrality in many important mental diseases. Furthermore, because of their neglect by logic and by psychology. There is, however, a likelihood that psychopathological methods will aid both logic and psychology. Somatic delusions do, it is true, afford some basis for a sensationalistic theory of delusions and indirectly of belief in general. But delusions affecting personality are perhaps better regarded as will-disorders or disorders of expression. At any rate, the writer's views were governed by his anatomical results in general paresis and in dementia præcox, which seemed to show that the majority of delusions were related to frontal lobe disorder. On general grounds the frontal lobes seem to the writer to be best regarded as organs for the elaboration of motion (including attitude, conduct, and the like). Of course the existence of essentially ideational delusions, here called fantastic, must be conceded: these beliefs are as it were *prima facie* delusions and do not require individual and specific testing in experience to determine their falsity. Such delusions were found in one disease (dementia præcox) related with parietal lobe anomalies or other lesions. However, the accuracy of the anatomical observations and their future confirmation are not essential to the argument. Nor is it necessary to consider the parietal lobes as an expanded and elaborated sensorium and the frontal lobes as an expanded and elaborated motorium in following these contentions. In point of fact, the pragmatic element in many or in all delusions is perhaps obvious to inspection, and the existence of a fantastic group of delusions, not requiring much pragmatic testing, is not unlikely on general grounds.

Assuming, then, for the moment that the value of comparing the categories of grammar with those of psychiatry is conceded

and that delusions have been chosen for a test of such comparisons, it becomes obvious that the strong motor, expressive, pragmatic element in delusions immediately invites comparison with the categories of the verbs.

III.

I am so ignorant of the theory of grammar that the present section of my paper must be very brief. At the outset I must perhaps say that the value of comparing categories of two sets of scientific data would be much diminished if those data happened to have been analyzed by the same group of men or under the same dominant logical interest. Had the theory of speech-function, language, grammar, and cognate materials been elaborated by the same technique as the materials of psychiatry, then the chances are that the comparisons here intended would be of lesser value. Luckily for these purposes, unfortunately perhaps for others, it would appear that the psychology which dominates philology and comparative grammar is not especially modern, and is indeed Herbartian. On the other hand, the development of aphasia doctrines and cognate matters in psychiatry has not considered to any extent the developments of philology, comparative grammar, or even the anthropology that has grown hand and hand with linguistics.

The ideas of Delbrück[1] about grammar and the ideas of Wundt about speech have undergone insulated courses. Steinthal and Paul seem to have been Herbartians, and Delbrück seems to have followed them. After Wundt's publication of large volumes on *Sprache*,[2] Delbrück brought out a little book of critique,[3] regarding many of the Wundtian contentions about speech as unwarrantable applications of personal and unproved psychology. Wundt replied in another small book.[4] There was no sign of unanimity.

[1] Brugmann and Delbrück, *Vergleichende Grammatik der Indogermanischer Sprachen*, 1886–1900.

[2] Wundt, W., *Volkerpsychologie, Eine Untersuchung der Entwicklungsgeschichte von Sprache, Mythus und Sitte*, I Bd., *Die Sprache*, 1900; H. 2, 2 Aufl., 1904.

[3] Delbrück, *Grundfragen der Sprachforschung, mit Rücksicht auf W. Wundt's Sprachpsychologie Erortert*, Strassburg, 1901.

[4] Wundt, W., *Sprachgeschichte und Sprachpsychologie mit Rücksicht auf B. Delbrück's Grundfragen der Sprachforschung*, Leipzig, 1901.

For our purposes this situation is on the whole advantageous, since we can trust the categorization of grammar to have proceeded without immediate and constant overhauling in the progress of psychology. Humboldt, Jones, Bopp, Grimm, Pott, Binfey, Schleicher, Brugmann, Whitney, and Delbrück himself are names of men hardly touched by psychology or logic. In fact the *Junggrammatiker* with their suspicion of metaphors in the whole range of their science would probably look on an incursion of psychology into philology as a genuine raid. They would probably recall with heart-sinking older efforts at a universal grammar, at a 'metaphysics of language'! There might indeed be a suspicion that somehow the psychological raiders were going insidiously to introduce still more deadly poisons into the already defiled wells of grammar than the 'bow-wow' or 'pooh-pooh' theories.

The present plan is more modest. Probably the streams of logic now current in linguistics and psychology parted as long ago as Kant. The categories of neither science have had much effect upon the other. Occasional references are made by expounders of the one science to the injurious effects of a possible resort to the other. Probably a 'nerve-brain' theory of linguistics would be regarded by philologists as hardly a degree removed from dangerous metaphors derived from 'natural' sciences, of which examples are cited especially against Schleicher. Giles says,[1] *e. g.*: "Schleicher and his followers in the middle of the nineteenth century had taken a keen interest in the development of the natural sciences, and had to some extent assimilated their terminology to that employed in those sciences. It was, however, soon recognized that the laws of language and those of natural science were not really alike or akin." Thus, by appeal to higher authority, are guarded the preserves of special theory.

However, on the other hand, in discussing these considerations with psychologists and philosophers, I find signs of an opposite tendency. A friendly critic remarked that he had always supposed that psychiatry and psychology could derive much aid from linguistics, in view of the obvious fact that thought and language

[1] Giles, P., "Philology," *Encyc. Brit.*, eleventh ed., Vol. 21, p. 431.

are so largely identical in mechanism. This contention was that in studying linguistics one is studying a branch of psychology and that in studying psychology one is nowhere or almost nowhere free from speech analogues. And, in the same direction, one is aware how much of the development of brain-localization theory in psychiatry is built up on analogies to the conditions prevailing in aphasia. The psychiatrist would here recall the efforts of the Wernicke school,[1] beginning with sensory aphasia and culminating in apraxia.

As against such contentions I find numerous objections to the employment of linguistic theory in the elaboration of logical and psychological doctrine. The logicians in especial seem aggrieved at the perverted usage of sentence-structure in syllogistic theory and are constantly calling attention to the pitfalls of language in respect to logic. Charles Peirce remarks[2] how much the logician Sigwart seems to depend on the expression of immediate feeling as logical, and how Sigwart considers language and especially the German language as the best vehicle of logic. It will be recalled how much attention is paid to 'substantive' and 'adjective' ideas in some of James's chapters. The reaction of most readers to the idea of 'but' or of 'if' runs, I suppose, to the effect that something figurative probably lies at the bottom of the linguistic analogy.[3]

We are often warned both by grammarians[4] and by psychologists not to trust overmuch to the situation depicted in Indo-European comparative grammar, *e. g.*, in the work of Berthold, Brugmann, and Delbrück. Thus the principles of the isolating Chinese, the agglutinating Turkish, the polysynthetic North American Indian languages are said to be impossible of establishment by means of terms borrowed from the Indo-European grammar.

[1] Wernicke, C., *Grundriss der Psychiatrie in klinischen Vorlesungen*, Thieme, Leipz., 1900, 2. Auflage, 1906, "Psycho-Physiologische Einleitung," S. 1–78.

[2] Peirce, C. S., "Modality," *Baldwin's Dict. Philos. and Psychol.* Macmillan, N. Y., 1902, Vol. 2, p. 92.

[3] James, W., *The Principles of Psychology*, Chap. IX, "The Stream of Thought," esp. pp. 243–8.

[4] Wheeler, B. I., "Language," *Baldwin's Dict. Philos. and Psychol.* Macmillan, N. Y., 1902, Vol. 1, p. 618, esp. 621.

Upon a superficial inspection of grammar we chose to believe that something of value to the theory of delusions, at all events to their nomenclature, could be obtained by a study of the theory of verbs in grammar. If the polysynthetic languages have no verbs, it is nevertheless undeniable that action is expressed by North American Indians. If incorporated languages often insert the object in the verb, yet at any rate the Basques are able to express action. If the Semitic verb has no tenses and merely expresses relations, yet at any rate there is a concept tense, which concept could be expressed by Semitic speakers. These examples suffice to hint at the great extent of the field of comparison.

I choose to study the grammar of verbs for the purpose of getting light on delusions or beliefs involving action. Much will be to the purpose, much not. In any event the grammatical nomenclature will not have been built up by psychologists or psychiatrists. We shall not identify grammar and psychology: we shall merely hunt for identities and analogies.

There is some indication that in Indo-European grammar there are four fundamental moods, imperative, indicative, subjunctive, optative. A discussion like that in Goodwin's *Greek Moods and Tenses*[1] exhibits some of the ingenious and appealing problems of these moods. Probably the germ of my desire to approach the present considerations was got from casual reading of the discussion by Goodwin of Delbrück's contentions concerning the subjunctive as a mood of *will* and the optative as a mood of *wish*.

The simplest verb forms seem to be the imperatives, bare stems as a rule. How readily these could be derived from cries, simple vowel calling, or at any rate simple articulations, early in man's development, can be readily imagined. The early world of the savage and the babe gets on to a considerable range of power with imperatives and the kindred vocatives.

Indicatives may then develop or, if not temporally prior to

[1] Goodwin, W. W., *Syntax of the Moods and Tenses of the Greek Verb.* Revised and enlarged. Ginn, Boston, 1890. Especially Appendix, "The Relation of the Optative to the Subjunctive and other Moods," p. 371–389, with specific references to Delbrück.

the subjunctives and optatives in verb-form development (and I suppose there are not enough comparative data from different linguistic groups to permit a general answer to such questions), then in any event logically prior. The world of language is full of statements, true or false, affirmative or interrogatory.

Figuratively presented, the linguistic verb stratum of imperatives is spread over with a layer of indicatives, which the increasing tranquility of life permits and produces. Imperatives and vocatives are less necessary, less polite, less useful, since past and future facts can now be held and turned over in the mind.

Gradually there may develop at the two poles of the language structure the moods of will and wish, to use Delbrück's terms. The development *might* of course be that, as a result of the operation of the fancy, the layer of the indicatives should be overlaid by a stratum of optatives, to which a number of *false* indicative statements might have made a convenient transition. Then further the layer of wishes *might* be topped with the layer of subjunctives, *i. e.*, of hypotheses, conditions, probabilities, and the like.

As we see men and women, however, I am inclined, for the present at least, to hold to the notion that the subjunctive and optative developments (of course always as mental reactions, *not* as verb-forms necessarily) take place rather independently. To be sure, the absolute deliverances of the *Utinam! Would that!* optative type do surely resemble imperatives rather than indicatives. And the more complicated machinery of a sentence containing a subjunctive immediately suggests the regularity and finish of the indicative. Both the subjunctive and the optative, however, have a derivative appearance and suggest the necessity of indicatives as at any rate logically prior to their formation. Hence, as above stated, I prefer to see the optatives and subjunctives rising as it were as separate eminences from the plateau of indicatives, and this despite the fact that special pipes may lead from the underlying imperatives to the moods of wish.

Perhaps I should here insist that the point of such a metaphor-

ical account of a certain aspect of verb-forms is not at all to offend any modern representatives of the *Junggrammatiker*. Above all, such an account has nothing historical or glottogonic about it. The point, if well taken, is logical not historical.

The student of human character and especially the alienist is at once aware that this fourfold division of moods (imperative, indicative, subjunctive, optative) fairly well corresponds with human character groups. Especially is this true of the subjunctive-optative contrast.

Who cannot see the scientific man as a man of hypotheses and probabilities, viz. of subjunctives, and the artistic man as a man of wishes and fancies, viz., of optatives? 'If me no ifs,' impatiently cries the poet to the man of science. 'The wish is father to the thought,' sadly or crabbedly the scientific man replies.

Such reflections as these, rather than genetic linguistic considerations, suggested the comparisons of the present paper.

More or less instructive comparisons between these fundamental moods and the classical temperaments might be made: thus, choleric, imperative; phlegmatic, indicative; melancholic, subjunctive; sanguine, optative. Probably the choleric and sanguine temperaments suit the imperative and optative moods more perfectly than do the others. There remains, however, something apposite in them all. It would not be difficult to show similar analogies between these four moods and the character types of Malapert, for example.

To sum up, at this point, after stating in Section I the *raison d'être* of these comparisons, the general reasons for choosing delusions as the *comparand* were stated in Section II, at the end of which section it was stated that the grammatical *comparator* must be from the region of the verbs. Section II had called attention to the pragmatic element in the majority of delusions, throwing this element into contrast with the ideational one. Some special reasons from brain physiology and from the writer's anatomical studies were adduced in explanation of the pragmatic element in delusions. These physiological and anatomical notions were not essential to the logical argument. But the fact

that somatic delusions seemed to crystallize about sensorial data (and were consequently rather more of the nature of illusions) and the fact that there seems also to be a second group of fantastic delusions (also more of a sensory nature and as it were illusions of memory and overplay of imagination) are two facts that tend, by the relative infrequency of their appearance, to emphasize the fundamental importance of the pragmatic element in most delusions. Most delusions are not *prima facie* false beliefs, but require the test of time and experience to prove their nature. This is but another way of stating their pragmatic, or at any rate their motor and expressive, character.

In Section III, a brief sketch has been offered of the situation in grammatical science, which seems to have developed along a path separate from that of the mental sciences, such as logic, psychology, psychiatry. The categories, nomenclature, and classification of grammar have therefore a certain independence from those of the mental sciences. Delbrück and Wundt do not gibe exactly. The section is finished by a brief statement as to the four moods (imperative, indicative, subjunctive, optative), which Indo-European grammar has shown to be fundamental. A figure of speech recalling the strata of geology is offered wherein the earliest practical situation in the development language is depicted as a layer of imperatives, next a layer of indicatives, and thereupon the subjunctives and optatives. Possibly these latter have a certain independence of development and spring from different parts of the plateau. The optative or mood of wish may possibly derive more particularly from the imperatives.

The next section will take up in order the most striking features in the categorization of the verbs which seem to be applicable to delusions.

IV.

Dismissing discussion as to choice of delusions as an object of comparison, and assuming that the pragmatic element in delusions is strong enough to suggest comparison with the most active and motor categories of grammar, I had proceeded in Section III to point out the independent development of the mental sciences on the one hand and grammatical science on the

other and to indicate in the briefest manner the characterological interest of the grammatical moods.

In the present section, I propose to rehearse some categories of the grammar of verbs that seem to me of theoretical and even of some practical value in the analysis of delusions. It is unnecessary to insist that the impetus to such comparisons is logical rather than psychological. It is not that thought and speech, pragmatic beliefs and grammatical moods, delusions and modal over-use or perversion, have developed *pari passu*. They may have developed *pari passu*, and speech may be as central in thought as aphasia is in the Wernickean psychiatry; but, if so, the point and origin of these comparisons did not lie in that identity.

Are there not logical categories ready to hand which are superior to any that may have developed in grammar? Notable is the fact that many logicians strongly condemn the grammatical infection of logical processes and the allied situation presented by the necessity of describing many logical processes in words. But, aside from the verbalism of much logic, let us consider a moment the logical modalities in comparison with the grammatical moods (or, perhaps better, modes).

There is a certain relation between the modalities of logic[1] and the so-called modes or moods of grammar. The distinctions of *possible*, *impossible*, *contingent*, and *necessary* are of obvious value in describing a variety of situations. In describing the actual facts that correspond to beliefs and delusions, these modalities are most exact. Or, if the 'actual facts' are not to be obtained, these modalities are of the greatest service in denoting what A thinks about B's statements, *e. g.*, what the alienist thinks is the truth about his patient's delusions. These modalities are of value in objective description. It is even possible to point out the vicinity of the concept *contingent* to the concept *subjunctive*, of the concept *possible* to the concept *optative*. It could almost be said that the *necessary* is not far from *imperative*. This would leave us with the *impossible* to

[1] Peirce, C. S., "Modality," *Baldwin's Dict. Philos. and Psychol.* Macmillan, N. Y., 1902, Vol. 2, p. 92.

correspond with the *indicative*, and perhaps, with the idea of Charles Peirce concerning the range of ignorance as corresponding with that of knowledge, some argument could be made even for the vicinity of the concept *impossible* to that of the *indicative*, In any case the *impossible* is well known not to be the opposite of the *possible*.

It must be clear from the comparisons here sketched that the classical modalities, *possible*, *impossible*, *contingent*, *necessary*, are of little immediate classificatory service for delusions or even for beliefs. Neither is there enough known offhand about any situation to make sure of affixing the proper modal description to the said situation, nor can the contentions of the believer or the paranoiac be subjected to experimental tests for the same purpose.

Accordingly, though the modalities of logic may be far more accurate and more representative of species of truth than the grammatical moods, yet the grammatical moods will perhaps prove more useful in immediate descriptions of belief-situations *from the point of view of the believer*, *e. g.*, of the deluded patient.

What we have long wanted in psychiatry is some way of getting at the psychic interiors of our patients. It is a safe injunction to hold fast from the first to the patient's point of view. The familiar Freudian distinction of manifest and latent[1] contents looks in this direction. But, omitting altogether at first any alienists' constructions as to latent contents, the examiner who adheres overtly to what is manifest in his patient's story is too apt, according to my experience, to fail to distinguish between what is true to the patient and what is true to the alienist. Let us distinguish what is *latent* in the patient from what is *manifest* in the patient. But let us distinguish between what is manifest to us *in* the patient from what is (to the best of our belief) manifest *to* the patient. Identical precautions are surely observable not only for patients but in the evaluation of all sorts of direct evidence.

One of the most valuable of the grammatical categories under which to consider a delusional situation or any belief-situation

[1] Freud, S., *Die Traumdeutung*, Deuticke, Wien, 1900.

in which the believer attributes a change in the universe is the category of the *voice*. Again it is important to distinguish the actual situation as the examiner views it from the situation as the patient or witness views it. We stick to the latter. Does the patient view himself as in the active voice, or in the passive voice, or perhaps in the middle (reflexive) voice? The question cannot often safely be asked in so simple a form. But it is as a rule singularly easy in a few questions to elicit from a deluded patient what he believes as to his own passivity or activity in the situation as he conceives it to be altered.

Perfectly simple is the felt passivity in certain victims of hallucination. The patients are here as passive as any recipients of sensation, and the whole reaction may be one of fixation or fascination *prima facie* passive. On the other hand, in cases of so-called *Gedankenlautwerden*,[1] the insistence of the hallucinatory or quasi-hallucinatory voices may be as intense, but is not necessarily one of felt passivity. The patient may be best described as in the middle voice: his conscience is at work, the still small voice is no longer small or still, he himself is somehow the source of his difficulty. Further reasoning may discover additional non-personal reasons or ancient active sins that are conceived by the patient to be actually responsible for the trouble. But this further reasoning is not necessarily faulty or in any sense delusional and may even be as objective as the alienist's own analysis. Indeed the patient may reason from manifest to latent as skilfully as the alienist or may even mislead the alienist by means of constructive or over-evaluated happenings of the past, which may then be taken falsely as actual objective happenings. And such constructions or distorted facts may prove new *points d'appui* for false beliefs. But the fact that this merry logical dance may be led both by patient and by examiner is not here in question. The point I am endeavoring to make is that the voice in which the patient's situation (to our best belief as to the patient's own point of view) can best be expressed is an important category of classification. Several alienists to whom I have submitted the point are in entire agreement with me and

[1] Cramer, *Die Halluzinationen im Muskelsinn bei Geisteskranken*, Freiburg, 1889.

regard the felt or conceived activity, passivity, or reflexivity of the patient as a surprisingly comprehensive characterization for the total situations presented by many deluded patients. That is to say, though it might be thought *à priori* that a given patient would rapidly shift in his deluded state from active to passive to reflexive (and permutably), yet the facts are commonly against these rapid shiftings of the felt 'voice.' Of course the phases do not always take so long in the evolution as in Magnan's *délire à evolution systematisée*,[1] now presented by Kraepelin in slightly modified form as *paraphrenia systematica*.[2] I shall not here enter special psychiatric questions; but limit myself to saying that in practice a given delusional phase in a patient is commonly well enough characterizable in a word as active (*e. g.*, certain states of delusional grandeur), as passive (*e. g.*, certain states of delusional persecution), or as reflexive (*e. g.*, certain states of self-accusation). The terms are good brief accounts of what I more cumbrously designated formerly[3] in such terms as 'ego-centrifugal,' 'egocentripetal,' 'spreading outwards,' 'spreading inwards,' and the like. Only the term reflexive is not so familiar and may need replacement with hyphenates of the term 'self,' or even with 'solipsistic,' 'egoistic,' though these latter terms are often too active in their denotation.

The fact that a situation may be described with correct grammar either in the active or in the passive voice need not trouble our analysis. So also can delusions. The point is not to identify grammatical voice with a type of delusional situation, but to borrow from grammatical categories a classification suitable for delusional situations.

Nor need a fact such as that in certain Indo-European developments the passive verb-*form* grew out of the reflexive verb-*form* be taken as of more than suggestive value. That fact might or might not be of telling value in such an analysis as ours.

[1] Magnan, "Leçons cliniques sur les maladies mentales faites à l'asile Sainte-Anne," *Gazette méd. de Paris*, 1877, and *Progrès médical*, 1887–1891. Also Magnan et Serieux, *Le délire chronique à évolution systématique* (Masson, Paris, no date).

[2] Kraepelin, *Psychiatrie, ein Lehrbuch für Studierende und Aerzte*, 8 Aufl., Bd. III, 1913.

[3] Southard, E. E., "Data Concerning Delusions of Personality. With Note on the Association of Bright's Disease and Unpleasant Delusions," *Jour. Abnormal Psychology*, Oct.–Nov., 1915.

Central in our considerations of the believer's active, reflexive, or passive voice is clearly the personality of the believer. We are thus naturally led to the possible comparative or suggestive values of the grammatical person. The grammatical concept and the common sense concept of person are to some extent obviously identical. The vast majority, if not the entire group, of psychopathic delusions may be said to revolve about the first person. The concept of the first person (singular) together with that of the voice synthesize to a concept which makes a fairly complete characterization of at least the majority of delusions. Delusions of grandeur as a rule readily reduce to the active voice and the first person singular: the predicate situations are often numerous and mutable. Delusions of persecution reduce as readily to the first person in the passive voice. Reflexive is the situation of the first person in delusions of self-accusation. Much of psychiatric interest doubtless awaits a grouping of other sorts of delusions even with so slight a logical armamentarium as this.

The second person is often involved in delusions. If we adhere to a projection of the delusional universe always from the patient's point of view, it must be clear how important is a distinction of second and third person. Taken from the psychiatrist's point of view, the *dramatis personæ* may well all seem to be in the third person, except perhaps the patient with whom the psychiatrist may feel like starting a small new drama of their dual own. But, if we adhere as ever to a construction from the patient's point of view, the difference between the *you* of the patient's plight and the *he* or the *she* may be decisive. Thus in minds working more or less on normal lines, it is hard to conceive homicidal ideas directed at a *him* or a *her*. The threats must far more often lodge with a *you*. On behalf of some *you* the patient might conceivably try to do to death a somewhat otherwise uninteresting *him* or *her*. But the majority of delusional situations are doubtless far more apt to be egocentric.

It may prove of special interest whether hallucinations of hearing come from a conceived *you* (as in a conversation or a monologue) or from a conceived *him* or *her*. There must be far greater intensity and dramatic quality about the statements of some *you* than from a third person.

It is entirely feasible to construct the situation of these other persons from the standpoint of grammatical voice. This has recently arisen in some cases that have come to my attention of *folie à deux*, in which the so-called 'active' and 'passive' persons may need separate analysis. And, in situations far less psychopathic, the psychiatrist has often to execute an about-face of this sort to get at the reactions of the grieved or angry husband or wife.

I have had to mention gender in the previous paragraphs. Krafft-Ebing and Freud have sufficiently called the world's attention to the sexual situations that occur in or make for psychopathies of various sorts. The routine collector of delusional elements must however bear in mind the necessity of establishing the sex of all the *dramatis personæ*, whether for the purpose of establishing or destroying some of the more recondite Freudian hypotheses or for the more modest purpose of banal social adjustments.

The value of the number of persons is not quite so obvious. How many persons are involved in the universe of belief or of delusion? Of course the scene may be peopled with any number of persons all acting normally even from the patient's point of view. But how many are acting abnormally either as sources of effect upon the patient, or as the objects of his action or perhaps as the instruments of his action? Are there perhaps some who may be fused and are working as a collective unit (the family, union-members, etc.) from the patient's point of view? Perhaps here is the weakest point in the routine analysis of delusional situations. The number of persons may be one, two, three, several, many, almost everybody, everybody, indeterminate, etc.; but all that can be collected concerning the number (and obviously the sex) of the persons involved, so far as the patient conceives them to be acting or suffering abnormally, will be found of the greatest value in analysis. Increase or reduction in the catalogue of intra-delusional persons may prove of value in prognosis. I should not need to insist on a special record of persons remaining extra-delusional, *i. e.*, excluded from the universe of the patient's altered world, when by all signs such persons would naturally be involved.

Most delusions of the lucid group which we can hope to analyze represent situations at least dyadic from the standpoint of the objective examiner. They are often triadic, *e. g.*, delusions of jealousy. But it must not be forgotten that a dyadic situation may conceivably be monadic from the point of view of the patient, as when he conceives that the altered attitude of a relative is not really injurious. But obviously enough there remains the suspicion that the situation, even from the patient's point of view, is effectively dyadic. Again delusions of jealousy may masquerade as dyadic.

Whether there is any important group of essentially tetradic delusional situations is worth inquiry. Among fictional situations as depicted by novelists, the tetradic situation with double shifting of courtiers is not unusual, though it may well be a more symmetrical situation than the world itself is apt to show. So far, I have not found many good instances of essentially tetradic delusional situations, *i. e.*, when the elements are persons. In numerous instances where four persons are involved, the fourth turns out merely ancillary to the third and to disappear, as it were, by the identity of indiscernibles. But this needs much concrete case analysis.

The important tense-distinctions of verb-forms recall the importance of the time element in delusions. Some of Delbrück's designations for general time relations of action are suggestive, *e. g.*, iterative, frequentative. Terminative actions, those conceived to have a beginning, an ending, or both, suggest obvious distinctions as to conceived delusional situations. Of course the stock case-history should and often does contain a sufficient account of these matters, as the term *history* insists. Still, I fear that we do not always keep separate in mind the objective anamnesis (to use a frequent medical term) and the anamnesis or catamnesis as the patient describes it and believes it to have occurred. Thus the one noxious event in the whole history may have occurred as it were aoristically at a special moment or brief period, and the rest of the history may seem to the patient an entirely natural train of consequences. In the direct or indirect psychotherapy, so apt to be employed in all

sorts of not-yet-defined delusions, quite a different technique might need to be employed for the delusional universe with an aoristic event long past than for a universe with iterative factors or with 'present perfect' characters, etc.

I arrive once more at the perhaps central topic of the moods. At the conclusion of the last section I spoke of the major distinctions as to moods, so far as the most thoroughly studied Indo-European grammar is concerned. I shall not in this paper deal intimately with the topic, as I conceive that much more case analysis should be available than I have as yet looked over.

But I wish to call attention to the vast wealth of special designations of moods which are found in the gradually increasing group of languages now being brought under scientific study. Most of these moods appear to me to fall rather readily into one or other of the subjunctive and optative groups. Thus the *conditional* certainly belongs with the subjunctives, and might perhaps be thought to offer a better general designation for the group. So too the *potential*. But *desiderative*, *precative*, *jussive*, probably belong with the optatives. As to the verb-forms and their special origin and appearance, the logician can have little to say. The point is, rather, that, if a verb-form exists to which a special name has been given, then at least some special shade of meaning has been thought to exist by the grammatical analyst. This shade of meaning probably expresses some rather concrete belief of *intra vitam* origin, not cooked up for a special purpose or at least for any psychiatric purpose.

I have more or less in hand a collection of these mood names from different grammars, of which a set probably large enough for these purposes is in existence at the Boston Public Library. The publication of the British and Foreign Bible Society[1] gives a convenient large list of languages, those in fact into which the Bible has been translated.

I hope to show, but will shortly dismiss here, the possibility that the transformation of 'subjunctive' beliefs into 'in-

[1] Darlow and Moule, *Historical Catalogue of the Printed Editions of Holy Scripture in the Library of the British and Foreign Bible Society*, Bible House, London, 1903, esp. Part IV, Indexes.

dicative' ones means paranoia of a pragmatic sort, whereas an identical transformation of 'optative' beliefs leads to delusions of the fantastic sort. 'Transformation' may be better rendered figuratively by such terms as degeneration, collapse, crystallization, condensation, degradation, etc.

V.

The object of this paper has been to illustrate the method of Royce's logical seminary at Harvard. No attempt has been made to describe the method, which is comparative rather than observational or statistical.[1] When the logician superposes the categories of Science A upon the material of Science B, or compares the categories of both, he is not at all sure of important results. If he obtains too extensive or too numerous identities by means of his comparisons, he may be compelled to decide that identity of categories means actual unity of materials. Thus, in the present instance, the reader may be the more ready to swallow the identity of certain categories in grammar and psychopathology, simply because he fundamentally believes in a larger degree of identity of speech and thought. In the event of such a nominalistic view as that, the only merit of the present essay would consist in spreading a sound method over new materials of the same sort; the method would not then be comparative in a very rich sense of the term. But, even if speech and thought are as closely allied as, *e. g.*, Max Müller thought them to be,[2] the fact still remains that the categories of linguistics and of psychology have not been wrought into their present form by the same group of men or under the same group of interests. If there is a partial identity of scientific materials, there is no evidence of identity of categories. The comparative method will then obtain a certain scope, even if that scope is limited to trying-out of special methods devised by linguists inexpert in technical psychology.

I hesitate to set forth the point; but I am left with a queer impression that linguistics falls short of representing logic in

[1] Royce, J., "The Principles of Logic," *Ency. Philos.*, Sci. I, Vol. 1, *Logic.* Macmillan, London, 1913.

[2] Müller, F. Max. *The Science of Thought*, Scribner, New York, 1887.

somewhat the same way that psychopathology falls short of representing psychology. I do not so much refer to the prevalence of concepts like 'phonetic decay,' 'empty words,' 'anomalism,' etc., in linguistics, although these concepts certainly suggest human frailty quite outside the frame of classical logic. I do not wish to construct a false epigram to the effect that linguistics is a kind of pathology of logic, attractive as this epigram might be. My point is that human facts are got at more readily in linguistics and in psychopathology than in logic and in so-called normal psychology.

For example, if I try to determine the logical modality of something and to affix the proper epithet (necessary, impossible, contingent, possible), I sink into a morass of factual doubts. But, equipped with the fundamental grammatical moods (imperative, indicative, subjunctive, optative), I can dismiss my doubts by describing them under one of these mood aspects, regardless of objective reality, truth to me, truth to Mrs. Grundy, or any situation except that depicted by the statement in question. The grammatical moods deal with evidence unweighed; the logical modalities require more weighing of evidence than is as a rule humanly possible. Psychopathology also deals with evidence unweighed. Particularly is this true of that portion of psychopathology which deals with false beliefs. Granted that some beliefs are *prima facie* fantastic and to us incredible. By the patient these fantastic and incredible beliefs are believed, but the nature and history of these fantastic beliefs may well be investigated to learn whether we are not dealing with a so-called wish-fulfilment (a Freudian technical term) or with a kind of degradation of what the linguist might term an optative attitude. But the majority of false beliefs are not *prima facie* fantastic and incredible. They on the contrary require the test of experience. They represent pragmatic situations. Granting the truth of certain hypotheses, we say, these beliefs might be accepted also as truth. Our thesis is that these pragmatic delusions do not represent a conceived wish-fulfilment, if by wish is meant a fancied situation. On the other hand, these pragmatic delusions appear to hang rather upon the degradation of a subjunctive

attitude, that is, upon taking as true a certain hypothesis. But neither fantastic nor pragmatic delusions can readily be classed under the logical modalities, *e. g.*, as possible or contingent, however possible and contingent they actually seem to the patient. In any event they are or will shortly turn out to be impossible, logically speaking, and, if the patient were to ascribe any logical modality thereto, he would be likely to deal in necessities on the one hand and impossibilities on the other. Grammatically speaking, the degraded optative belief may even set into an imperative, and beliefs degraded from both the optative and the subjunctive appeal to the patient as indicative, if not yet imperative.

From our superficial study of the categories of grammar as they revolve about the verbs, we have come upon two considerations of value that are not entirely obvious, the psychopathic analogue of the grammatical 'voice,' and the question of two main types of delusion degraded respectively from 'subjunctive' and 'optative' attitudes.

I believe that the 'voice' distinction will forthwith appeal to all psychiatrists as valid within its range. The distinction seeks to express the relation between the world and the individual from the individual's point of view under two forms, (*a*) that in which the self is active and (*b*) that in which the self is passive in relation to the environment; but in the third place (*c*) the relation of the individual to himself is suggested, viz., under the 'middle' or reflexive relation. Whether the reflexive relations of the self break up further into a group where the 'I' dominates the 'me' and another where the 'me' overpowers the 'I' (that is, whether the ego is sometimes active in respect to itself and sometimes passive), is a question partly of fact, but more of the nature of the self and of the whole difficult topic of self-activity.

Whether the distinction between pragmatic delusions (as it were, precipitated subjunctives) and fantastic delusions (as it were, precipitated optatives) is valid, must remain undetermined. The distinction has at least the value of suggesting a similar distinction in human character in general; both distinctions may be derived from identical psychological facts.

If in the practical handling of a patient, or indeed of anyone else in a situation hard to interpret, the observer can make out the 'voice' of the subject's situation from the subject's point of view, and can secondly determine whether the difficulty rests upon trouble with hypotheses or trouble with wishes, much is gained surely.

We saw also from our incidental study of person, number, and gender how important might become the question of monadic, dyadic, triadic, or polyadic situations involving false beliefs. The collection of groups of such situations for analysis is certainly indicated, naturally with invariable reference to the 'voice,' active or passive, of the patient or central figure. Fiction and drama could throw some light on these matters.

In the gathering of data for analysis, it is clear also that the time-relations must also be studied from the patient's point of view, to the end of determining whether the particular subjunctive precipitate has relation to some central point in the past, whether the particular optative precipitate has relation to a present or present perfect situation, or whether other 'tenses' come in question.

E. E. Southard.

On Descriptive Analysis of Manifest Delusions From the Subject's Point of View

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ON DESCRIPTIVE ANALYSIS OF MANIFEST DELUSIONS FROM THE SUBJECT'S POINT OF VIEW*

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(*Read in abstract at the Washington meeting of the American Psychopathological Association, 1916.*)

I fear that the present paper may be regarded as an elaboration of the obvious. I am, however, the more content with such a view of my analysis in that the psychiatric world of late has been invited to much that is far from obvious. The term 'manifest' in my title suggests that the so-called 'latent' in so-called 'mental mechanisms' is not here to be 'analyzed,' if indeed it be susceptible of analysis in the classical usage of the term. Likewise, the term 'descriptive' in my title indicates that no claim is made to 'explanatory' analysis, if indeed analysis (in the classical sense) ever did 'explain' anything. In short, if the descriptive analysis of the manifest in false beliefs here meant turns out to be a valuable preliminary to work on 'mental mechanisms,' I shall have no objection. But I do feel that so-called 'psycho-analysis' or any other so-called 'analytic psychology' which begins to synthesize (*e. g.* to symbolize) from the outset is more likely to import the examiner's own beliefs (true or false) into the particular psychopathic situation than to extract the patient's beliefs therefrom. And this remains true even if the patient's beliefs are not the *primum movens* of his total attitude or behavior. For,

*Being M.C.M.D. Contribution, whole number 150 (1916.8). The previous contribution was by Dr. Herman Adler, entitled "The Cholesterol Content of the Blood in Psychopathic Patients," read before the Association for Clinical Investigation at Washington, D. C., May 1916.

granting that *aliquid latens* is actually at work, it is important to know also what the subject thinks is at work. Surely the subject's thoughts about whatever is manifest to him modify his attitude or behavior to some degree, and form at least a part of his rationalization thereof. If the total process of reasoning is not 'conscious,' surely *some part* of the reasoning process *sometimes* s conscious and employs cognitive factors.

I attempt to deal in fine with beliefs and delusions (a) that are *manifest not latent*, (b) by a process that is *analytic not synthetic*, (c) to the immediate end of *description not explanation*, and (d) from a point of view that is *subjective to the believer* and (so far as possible) not subjective to the examiner and without pretense to being objective as the total account of a psychopathic situation.

The attempt is in no sense a critique of analytic psychology and in fact grew out of practical necessities in the clinic of the Psychopathic Hospital in Boston where a stream of internes and assistants, medical and social, flows in and out without particular previous instruction in psychopathology or compensatory knowledge of the world. The prime necessity here was to supply captions, compartments, items indispensable to the proper analysis or later synthesis of a given psychopathic situation. Faced with such a situation as presented by a perfectly lucid patient, the tyro in psychiatric examination is embarrassed by riches of information, by a luxury of woe, which at first seems infinite in dimensions, perhaps hopelessly tangled. Conflicting accounts by the patient, by one or more parties in the patient's *entourage*, by public or social agencies, by previous physicians, to say nothing of the prejudice of first impressions by the examiner, combine to confuse the very elect. The examiner's possible preconceptions that "*nothing manifest is at all likely to be the 'real' explanation*" makes confusion worse confounded. The examiner dashes after Ariadne's thread without due consideration whether or no there is a labyrinth at all. What therefore, I asked, was the indispensable minimum of items required for orientation in a patient's seemingly (to him) altered, seemingly (to us) delusional world?

I offer below a list of such items in an orienting analysir of seemingly false beliefs. The process by which the list was arrived at seemed to possess intrinsic interest and is therefore described, although the value of the items depends in no wise upon the technique of the'r choice.

The fact that the items are as ancient as the foundations of grammar is of some interest. The descriptive bio'ogist in his capacity as behaviorist might well seek for the subheadings of his descriptive science in logic itself. Studying as are we a subjective situation rather than primarily an objective history of actual events, it was perhaps natural that grammar and rhetoric rather than logic should supply suggestions for descriptive headings; e. g. (and see also below) what seems necessary to the patient *is objectively not "necessary," but it remains "imperative."*

I have been able to clarify at least my own mind by resort to some of the more obvious categories of grammar for the purpose of analysis of delusions. I feel sure that several of the distinctions made will appeal at once to the majority of psychopathologists as they have appealed to a number of my colleagues in practical work. The categories chosen are, in fact, so many thousand years o d, that they cannot fail to be of some value, as I think will appear on inspection of that division of verb theory dealing with person, number, gender, and tense. That is to say, it must be obvious to the layman, to say nothing of the psychopathologist, that it is important to know who inhabits the universe of the patient's false beliefs, how many persons are involved in the delusional universe, what the sex of those persons is, and when and for how long the noxious event or condition is thought to have occurred or lasted. Indeed it may be regarded as a fact, or at any rate as a pious wish, that all proper histories of patients contain enough upon which to ground a judgment as to person, number of persons, sex, and time in the alleged delusions. If grammatical categories are of any special value here, it is only that they give us a certain sense of completeness as to possible items of evidence relative to false beliefs. Clearly enough, the tyro in psychiatric examining often does not know how far to go in the taking of evidence, and rarely ends taking a history without

a gnawing sense that he might well go infinitely farther in securing testimony. Accordingly, I had for some time been seeking some convenient termini for history-taking to which a tyro might safely pin his faith. I find that the grammatical items just listed, not only satisfy the tyro and give him a sense of relative completeness in examination, but they can also serve fairly well as a basis for more elaborate examination.

In addition to these obvious items (person, number, gender, tense), which might as easily have been developed from anybody's inner consciousness as from a review of grammatical categories, I wish to call attention to two distinctions of equal interest but of somewhat more doubtful value, and at any rate of far less obvious derivation from the facts as the patients present them. The two categories in question are those of the grammatical "voice" and of the so-called grammatical "mood," or mode. Before developing what I consider to be the values of the categories of voice and mood, let me repeat that the kind of analysis I wish to support in the first instance is analysis directed at what may be caelld the manifest rather than the latent aspect of the psychopathic situation. I do not mean to say that ana'yses, itemized as I here suggest for the manifest, would not be equally suitable in the realm of the latent. My plea would be for an *analysis of the manifest in the delusional universe prior to that of the latent.*

Let us turn to the grammatical categories of voice and mood. First as to the category of the voice, with its subdivisions, active, passive, and middle (or reflexive). I learn that my colleagues find the grammatical voice condit on as it were to "click into place" in their analysis of a great many delusional situations. In our difficult combination of extensive and intensive work at the Psychopathic Hospital in Boston, where we deal with a great many cases that are not obviously insane or certainly psychopathic, we have naturally developed a technique of examination *more Socratico.* It is clearly not advisable as a rule to ask a patient whether he is in the active voice toward his environment or whether he is passive therein; and it is clearly far from likely that the patient would understand being placed,

as it were, in the middle voice. Nevertheless, it is surprisingly easy to develop what the patient believes as to his active or passive relation to the environment, and this by means of a very few questions. It certainly takes far 'ess than the proverbial "twenty questions" to determine whether the patient is manifestly and subjectively in an active and dominant relation to his fellows, in a passive relation thereto, or in a personal plight of difficulties with himself. Mixtures of these relations also occur. Nevertheless, t is surprisingly often the case that the total situation conceived by the patient as altered is one to which one of these three categories of the grammatical voice—active, passive, and reflexive—may be given. It is plain that now and then a patient regarding himself as dominant in his environment may assume a passive attitude, as of one in ambush or playing 'possum, or on the principle that "still waters run deep," and the like. It is likewise plain that a patient regarding himself as overwhelmed by his *entourage* may become counteractive to somebody therein whom he takes to be a special foe. In these instances of the subjectively-dominant-person-playing 'possum, and the subjectively-overwhelmed-person-counteractive, we are undoubtedly dealing with *objectively* passive and *objectively* active persons. Subjectively, analysis promptly shows, the patients in question are, as it were, *actively playing 'possum*, and on the basis of being overwhelmed, as it were, *passively counteractive;* that is, active in the capacity of a victim. It would seem a contradiction in terms to speak of the proverbial caged beast as passive; objectively the beast is as mobile as you please; subjectively he is full of feelings of effort, etc., but he is, nevertheless, both objectively and subjectively in the passive voice grammatically speaking. Suppose, now, we are confronted with a patient feeling, as so many feel, "cabin'd, cribb'd, confin'd," despite the objective absence of such bonds, is it not wise to regard the responsive activities of the patient as quasi normal, namely: as not (from the patient's point of view) essentially other than what a normal person would do under the circumstances? The fact that the circumstances are delusional

does not render them any the less credible and credited by the patient.

I think it may be at once recognized that there are certain values attaching to the statement that the patient is in a general way in the active voice or in the passive voice with respect to his environment. It is not so certain that the category of the middle voice is equally valuable. I find that the middle voice again splits up into two; that there probably is an active and a passive form of the reflexive or middle voice. There are probably at least two sorts of internal psychopathic situations which may be characterized by the term *reflexive*. It is, of course, probable that there are two sorts of moral situation in general which comport with this distinction. For the present, however, I am insisting merely upon the value of these distinctions on the psychopathic side. I would make employment here of the dist nctions between the forms of self so beautifully described by James in the chapter on the consciousness of self in his *Principles of Psychology*. He there speaks of the following three kinds of self (I omit 'pure ego' from the present discussion): the *spiritual*, *material*, and *social selves*. Dismissing for the moment the social self as having to do largely with the relations of the subject to his environment, I would consider especially the spiritual and material selves, or "ego" and "me" of James' nomenclature. Just as the patient's relations to his environment may be formulated as follows:

(a)	Active	PAT>ENV
(b)	Passive	PAT<ENV

so we may consider that the patient's relations to himself may be formulated somewhat as follows

(c)	Reflexive	(Active)	EGO>ME
(d)	Reflexive	(Passive)	EGO<ME

The sign > in this formula obviously means many things. Thus, a patient active with respect to his environment (PAT>ENV) may be a patient with delusions of grandeur

giving a variety of orders to his *entourage* or he may be violent and destructive in his environment. He may even, as was pointed out above, inhibit his objective activities for the moment to secure an ulterior end. The (grammatical) activity includes an almost limitless number of forms of action. The point lies in the patient's conception of his relation to the environment. So also with the sign $<$ A total gamut of relations may be covered by this sign, from the extreme instance of the counteractivities of the caged beast to the more ordinary phenomena of the ordinary delusion of persecution. When we come to the relations of the patient to himself, to the reflexive disorders, our difficulties multiply on account of the well-known logical pitfalls of identity and non-identity. It is not necessary to read Hegel to become aware of the difficulties of the concept 'self-activity,' and the concept of 'self-passivity' is not less involved. Just as a normal subject fits his environment and the environment fits him approximately, so normally there is a similarly satisfactory relation between the various parts or categories of the self. We speak of self-control, of being at peace with oneself, of having settled one's own problems, of the serenity of virtue, and the like. These are examples of perfect fit between the spiritual and material selves. The doctrine of humility which prevails in Christianity possibly preaches what the youth believes to be too great a degree of self-abnegation or passivity. It is possible that our current idea of self-control is a little more that of the Christian self-abnegation than that of the strong man having himself well inhand. However that may be, it is safe to say that psychopaths often show degrees of deviation from what may be called the standard reflexive relation of the self to itself which are entirely convincing, and illustrate both forms of altered relation of the self to itself, namely: the active and the passive forms of reflexive disorder. What the French term *theomanias*, or forms of disease with religious exaltation occasionally show the spiritual self in entire control of the bodily self. Psychopathic degrees of flagellation may be used as examples of this kind of disorder. Certain delusions of grandeur of a self-contained type of egoism may also illustrate the form.

The passive form of reflexive condition is illustrated by a number of conditions ranging from the feeling of inadequacy of the depressed form of manic depressive psychosis, masturbation, and the like, up to certain forms of suicide.

Enough has been said to suggest briefly the advantages of employing the 'voice' distinction. The tyro in psychiatry—and we may all lay claim to this title as we confront many of our problems—finds a great number of his cases to click into place with the use of this four-fold system of relations as formulated above. A sympathetic harmony with the patient's own attitude is attained by the examiner, whose questions become rather legitimately leading questions, the answers to which speedily betray any loss of dramatic unity which would be endangered by the patient's slipping from one of these four relations to another. The latter, indeed, happens in sundry cases of schizophrenia. If we cling, in our analysis of the lucid patient's beliefs, to what is manifest and subjective in the patient, and strive to understand the patient's behaviour from one or other of these four relations, I believe a certain insight is gained which is the best preparation for more thoroughgoing factorial analysis, and for the syntheses of successful genetic work.

I turn to the equally interesting but more dubious region of the grammatical moods. At this point, I may interject an explanation of my looking in the direction of the moods of grammar for psychopathological categories. As I have set forth more in detail in an article written in honor of Prof. Josiah Royce for the *Philosophical Review*. I had occasion to illustrate the method of Prof. Royce's logical seminary by comparing the facts of one science with the classification and nomenclature of an older science. For reasons which need not concern us, I chose to compare psychiatry with grammar. I had been trying to come to clearness for myself as to the topic of delusions. Finding that many, if not the majority, of delusions are not perverted ideas so much as perversions of the believing process, I was convinced that I should find more to my comparative purpose in the grammar of verbs than elsewhere. Looking through Goodwin's *Greek Moods and Tenses*, I came upon traces of

the old controvesy about the true nature of the subjunctive and optative moods. I found, for example, that Delbrück held that the subjunctive was a mood of will and the optative a mood of wish. Knowing that much had been made of the term *wish* by several workers in the field of psychopathology, and feeling that over rationalization of the subjunctive type was the rule in other camps, I became persuaded that much might be learned from the situation in grammar of possible value in psychiatry. Not knowing much about grammar, I was *à priori* not very likely to get very far in correlations.

However, without further consideration of my reasons for effecting such correlations, I will proceed to psychiatric suggestions drawn from the moods. It is often stated that the term *mood* is itself not so good a term as the term *mode*; exactly why the Latin term *modus* should be interpreted in English by the emotion-laden term *mood*, I am not competent to discuss. Strangely enough, however, the English term *mood* so well expresses a certain form of rhetorical situation that every educated person would clearly understand being put in, *e. g.*, the imperative mood, or perhaps the optative mood. It would be less idiomatic to speak of a person as in the subjunctive mood, although a person mulling over hypotheses would probably be the connotation of such a phase. A phlegmatic or matter-of-fact person might be described as usually in the indicative mood without undue stretching of terms. At all events, these four moods: the imperative, indicative, subjunctive, and optative, are apparently the four characteristic moods or modes of the best studied languages, namely; the Indo-European group. Other languages contain a variety of other variously designated moods, but we find these moods reducing as a rule to the standard four. In fact, the majority of fresh designations, such as the potential, conditional, and the like, on the one hand, and the desiderative, precative (prayerful), or jussive (statements of *lux fiat* type), moods, seem to flow in the direction of the subjunctive or the optative, as the case may be.

Without going into this matter at length or justifying the idea by historical data I may briefly say that these

moods may be logically related to one another as in the following diagram of the hypothetical development of moods:

Subjunctive Optative

Indicative

Imperative

I have developed in my article for the *Philosophical Review* some notion of the layered development of these moods, pointing out that the child of the savage may well start with those bare stems that constitute the imperatives; that upon the layer of the imperatives may develop the matter-of-fact indicatives; and that upon this plateau, the two eminences of the subjunctive and the optative develop. Science is a matter of hypothesis and employs subjunctives. Art is in part at least a matter of imagination and flourishes upon optatives. For *a* certain type of mind, the wish is father to the thought; optative air-castles secure the subject's happiness. The scientific or hypothetical type of mind attempts to realize itself, as it were, subjunctively. The optative person builds, as we say, castles in Spain; the subjunctive type is gulled by the well-known 'Spanish captive'. hoax

It is a curious thing that of the anciently accepted temperaments, the choleric corresponds somewhat closely with the imperative The phlegmatic may be said to correspond somewhat with the indicative; the sanguine rather clearly with the optative. Whether the melancholic or atrabilious corresponds with the rationalistic employment of subjunctives is not at present wholly clear to me. The correlation between the temperaments and the moods is sufficiently close to be at least suggestive.

The logician might inquire why we resort to grammatical moods when we have the logical modalities at our command. These modalities, as is well known, consist of the *necessary*, the *contingent*, the *possible*, and the *impossible*. It is clear that the objectively necessary corresponds somewhat closely with the imperative. It is clear that the objectively con-

tingent and the subjunctive are closely allied. The possible and the optative are a so not far removed from one another. The relation of the impossib e to the indicative is not at first sight close, and only becomes so when it is developed by the logicians that the impossible is not so much the opposite of the possible as it is the opposite of the necessary; and that just as the imperative and the indicative are coupled together somewhat apart from the optative-subjunctive couple, so the necessary-impossible couple is a little apart from the possible-contingent couple. Here again, the correlation of the four-fold systems: logical modality on the one hand, and grammatical mood on the other, is perhaps not exact, though it is decidedly suggestive.

As against a logician who should decry the use of the grammatical moods instead of the logical modalities, I would insist as above stated on the preferential use of grammatical categories for subjective situations where the truth may never be learned, and where, beliefs that we regard as false, the patient regards as true and upon which he proceeds to act accordingly. It may then be urged that the employment of these mood designations is an approximately exact way of expressing inexact situations.

Practically, then, I find my colleagues, although they do not accede to the mood distinction as speedily and, as it were, passively as they do to the use of the voice distinction, to some extent adopting the mood distinction. My own idea is that the degeneration, condensation, collapse, or precipitation of a subjunctive into an indicative;—that is, of an hypothesis into what the patient regards as a fact,—leads to a mental situation of a pretty definite complexion. Signs of pre-existent hypotheses are frequent. Elaborate argumentation is the rule, as after all the hypothesis makers are precisely the eager disputants of the world. On the other hand, when the optative degenerates, condenses, collapses, or precipitates, then again one finds evidence in the resultant indicatives of the pre-existent wishes. In the former case, the subjunctive precipitate is in the form of what may be termed a pragmatic delusion or a para-pragmatic belief, namely; a false belief which requires experience to determine its falsity; whereof we say, "Time

will tell." On the other hand, the optative precipitate is in the form of fantastic delusions which are, as it were, *prima facie* false, that is, false taking into consideration the context and circumstances of the patient. These latter delusions should not require the test of experience. They are not irrational beliefs or rational delusions; they are paraphantastic beliefs or fantastic delusions. I offer as a suggestion, therefore, the conception that *delusions may be descriptively classified as degenerate hypotheses, on the one hand, and as degenerate wishes on the other.* If any one desires to identify hypotheses and wishes, I shall naturally have no objection when proof is brought. The descriptive classifications of delusions would fall together at precisely such time as the distinction of hypotheses and wishes should vanish. I make no point of the ultimacy of the distinction. Whether it would not be possible to divide the characters of men along these lines, I shall not develop here.

So much for a brief statement of certain categories deliberately derived from grammar that may be of some use in the psychiatry and especially in the psychopathology of delusions. I believe that it is clear that the analytical items of person, number, sex, and time, as involved in the noxious events or conditions of the delusional universe, must be of value in description. I believe that the four-fold system of possible relations of the patient to the environment, on the one hand, and of the patient to himself, on the other, each relation splitting into a pair, active and passive, will also more or less appeal to the analyst. The suggestions from the grammar of moods are somewhat more doubtful, but, to say the least, suggestive.

In a given case, how much to the point it may be to ask what degenerated hypothesis is at the bottom of this irrational belief, or on the other hand what precipitated wish; again, how important it may be to ask whether this patient from his own point of view is dominant in his situation or overwhelmed by it; or whether, on the other hand, his difficulties are intrapersonal and relate to disorders in the relative values of his different selves; whether he is in the seventh heaven of neglected bodily concerns, or whether he is hypochondriacally controlled by somatic factors. Again, is

the first person involved alone, or are two persons involved; and is the other person involved in the dual universe conceived as in the second person, namely: as a *you*, or in the third person, as *him* or *her*? The dramatic situation is entirely different when one is *vis-a-vis* and when one is an eavesdropper. Is the situation not monadic, not dyadic, but triadic? Is this a jealousy situation, representing the so-called triangle of the novelists; or is the triangle situation always actually, as a colleague suggests, essentially tetradic in that another model is being more or less unconsciously utilized, upon which to build the actual triadic jealousy situation? All these and numerous other intriguing problems develop in the form, either of hypotheses or of wishes, on the part of the psychopathologist.

Summary

The writer aims at a descriptive analysis of manifest delusions and false beliefs taken subjectively *i. e.* from the patient's point of view. He regards this as an indispensable preliminary to explanatory synthesis of psychopathic situa tions, even should it turn out that *aliquid la ens* is the nucleus of such situations. Practically he proposes a minimum of terms which the tyro in psychiatric examination must aim to get from a lucid patient entertaining or alleged to entertain false beliefs. In addition to (a) The *person* or persons involved, (b) the *number* of persons involved, (c) the *sex* of these persons, (b) the *time*, past, present or future, in which the noxious event or condition is believed to occur, the writer deals also with (e) the 'voice' in which the patient takes himself to be. The patient from his own point of view regards himself as at odds with the environment

(1) as it were actively
(PATIENT>ENVIRONMENT)

or (2) as it were passively
(PATIENT<ENVIRONMENT), or again as at odds with himself, either

(3) with higher (spiritual) self dominant
(EGO>"ME")

or (4) with lower (material) self dominant (EGO<"ME"). The writer deals also with (f) the distinction of 'mood,' finding that patients above the 'imperative' level entertain either irrational delusions or fantastic ones. The writer speculates that irrational (pragmatic) delusions represent hypotheses taken as facts (*i. e.* 'subjunctives' degenerating into 'indicatives') and that fantastic (*prima facie* false) beliefs represent wishes taken as facts (*i. e.* 'optatives' degenerating into 'indicatives'). Possibly those who transcend the imperative and indicative levels in normal development split into two classes of persons, those with a leaning toward hypotheses (highest development, men of science) and those with a leaning toward wishes (highest development, artists). In the body of the paper some account is given of the comparative method by which these items of psychiatric analysis were obtained, a fuller account of which has appeared in the *Philosophical Review* in a paper written in honor of Professor Josiah Royce.

[1]Southard: On the Application of Grammatical Categories to the Analysis of Delusions; article read in honor of the sixtieth birthday of Professor Josiah Royce at the Philadelphia meeting of the American Philosophical Association 1916. Published in the Philosophical Review May, 1916.

ZONES OF COMMUNITY EFFORT IN MENTAL HYGIENE.*

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Dr. Copp set me the question "How Should the Community Be Organized for Mental Hygiene?" I intend to circumscribe the topic for the present by considering chiefly the aspect of institutions and agencies of a public or social nature. I shall not wander into the more attractive but remoter fields of constructive effort in mental hygiene of the future. I shall point out the desirability of having the blood circulate freely in the periphery of the institutional network, as well as in the executive heart. I shall try to utilize a bit of the war nomenclature, by contrasting the zone of advance in social work (which I take to mean the schools, courts, dispensaries and other public and social agencies coming in direct contact with the people), with the zone of the social interior, so to speak, (namely, the hospitals, schools for the feeble-minded, reform schools, prisons, placing-out systems, and the like, which the community has long recognized as valuable and inevitable mechanisms). As somewhere on the line of communication, between the zone of social advance and the zone of the interior, I shall speak of the psychopathic hospital which has the comparative task of distributing doubtful cases that cannot be diagnosticated at first sight and which require more protracted analysis.

But before I come to talk about the zone of advance, the line of communication and the zone of the interior, socially speaking, I shall say some words about the general principle of community organization which I shall hope to show must be managed on democratic rather than on autocratic lines. I shall commend the application, as a social principle, of our motto *e pluribus unum,* commending the *e pluribus* type of unity as against any "hand-me-down" type of *Kultur.* I shall point out how effort should begin in the districts rather than in the executive centers and how a community will achieve a complex institution like a psychopathic hospital only when the zone of social advance is ripe therefor. I shall then speak of a little problem in the organization of our own mental hygiene in this conference,—but more of that later.

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*No. 120 Reprints of Reports and Addresses of the National Conference of Social Work, 1917 Meeting at Pittsburgh. Order by number. Write for descriptive list of publications, 315 Plymouth Court, Chicago, Ill.

Being contributions of the Mass. Commission on Mental Diseases, whole number 193 (1917.13). The previous contribution, No. 192 (1917.12), was by E. E. Southard, entitled "The Desirability of Medical Wardens for Prisons," in *Proceedings* of National Conference of Social Work, 1917.

What I shall say about organization for mental hygiene will be interlarded with remarks on autocracy and democracy. There are many of us here who are strong believers in unity of purpose, in unified socialization (as the phrase goes) of human interests, and some of us yearn for the unity of purpose which autocracy confers. Some of us, good Americans though we believe ourselves to be, are yearning for that socialization—"made-in-Germany"—which a beneficent (if not benevolent) despotism could so rapidly contrive. I must confess I am not one of these. Unity of purpose I want, but not the unity that autocracy hands down. In point of fact, unity is just as often of democratic as of autocratic origin. And the soundest form of unity is what may be called the *e pluribus* type.

I suppose there is no wiser step ever taken by our democracy in adopting the literacy test for immigrants. There was an example of democratic vision that took decades to mature into clear conception of law. A benevolent despot might have conferred it upon us long since—our elective despots never did favor the measure.

Mental hygiene must begin at the bottom, in the district,—not at the top, in the Union as a whole. Take that powerful and splendid organization, the National Committee for Mental Hygiene. True to Anglo-Saxon type, of course, the National Committee is perhaps a bit local in its management, a bit Manhattanized, as a mere Bostonian speaking in Pittsburgh may safely hint. But benevolently and beneficently the National Committee proceeds on its way, by state survey and county survey, by the encouragement of ever more local committees, towards the ultimate development of a national ideal for mental hygiene.

But, I insist, the *e pluribus* principle of unity must prevail here also. Massachusetts methods would not fit Arkansas. More surprisingly still, they perhaps would not fit New York. The mental hygiene propaganda does not work like a rubber stamp. Are we not here repeating history as made in the field of social service at large?

How, then, should the community be organized for mental hygiene? The phrase "community" strikes me as too large, as suggesting the beneficent, up-to-date rubber stamp descending from above. *E pluribus* unity, a distributive-towards-collective, not a collective-towards-distributive evolution, a hygiene beginning in the commune, district, or local unit,—this seems to me the distinctive necessity of this particular progress.

Even if it were not so, even if we possessed superstate or supernational principles of mental hygiene ready to apply, yet the very inequality of progress in the various states at the present time would halt our efforts at organization of a *Great Society* like that of Graham Wallas, or the "blessed community" of my master, the late Josiah Royce.

I have sometimes thought a trick could be turned in some great mechanism for mental hygiene such as the *Saturday Evening Post* by

contributing a screed, "On the Degrees of Civilization in the United States"!

The fact that there are degrees of civilization amongst the states of the Union is a fact that in itself negates the idea of organizing the country for mental hygiene by any of the rubber stamp methods so pleasing to the constructors of "programs," "pleas" and "plans" in the beloved committees of the great organization societies. I doubt whether such rubber stamp organization would work even in a homogeneous community, were such conceivable. But in any event, there is no social homogeneity, neither homogeneity of sections nor of states nor in many instances of the counties themselves. Of course one of our most ancient heritages is the county or commune division. That the county system sometimes solidifies into evil mechanisms such as "county rings" and the like, I will not deny; but the point remains that the unity of social management, whether in mental hygiene or otherwse, is far below the level of the federal unity or of the state unity. It is not that mental hygiene, like charity must begin necessarily at home, but surely it must begin in the district and not in the cold marble capitols where executives wield their rubber stamps.

All this may be taken somewhat impatiently by the social worker who is aware how fundamental is the neighborhood and settlement principle in modern social service, and the inadequacy of the executive seal may be accepted as a truism by social workers who have dealt with some state agencies. Yet I suspect that many of the chiefs of social service here today, confirmed though they are as to the value of neighborhood work, entertain in another corner of the mind the vague thought or desire that right organization will somehow, some day, be conferred from above.

Experience in the mental hygiene branch of social service or the social service branch of mental hygiene—for the two fields, overlapping widely, are each provided with vast reaches of service hardly touched by the other—has proved the value of proceeding *e pluribus* to unity. I can speak with familiarity only of such a highly wrought social situation as faces us in the metropolitan district of Massachusetts. In Massachusetts, as in several other densely populated states, the progress of social organization (I am of course here referring to non-public, non-governmental organizations) has been to unity. Other states, now beginning to urbanize and to present the social complexities of the older states, may desire to adopt out of hand the experiences of these older states. As for mental hygiene, it might be conceived that a state board for mental hygiene could somehow introduce a consciously and shrewdly conceived system for, let us say, Arkansas, which would in due time bring the Arkansans as reasonably near millennium as we can now imagine. But such a scheme would hardly be more *a propos* than the Teutonic scheme of rubber stamping *Kultur* upon the world at large. Just as Germany feels that the mental hygiene of Alsace-Lorraine is

better in German hands than in French or in the hands of the inhabitants themselves, so the social organizers of our commnuity are apt to feel that a rescue must be made of, for example, Arkansas, Texas, or even Illinois.

Mental hygiene organization, like charity organization, begins with the district. It becomes more imminently necessary in highly urbanized districts under modern metropolitan conditions. Here, to use a military phrase, is the *zone of advance* in the *theatre of* mental hygienic or social service *operations*. Social institutional devices for mental hygiene should, to my mind, be placed in this zone of advance. Aside from the refinements of the psychopathic hospital and elaborate social service centers, and aside from the strategy of organization societies and the executive rubber stamp, there should be in the zone of mental hygienic advance mechanisms for early and immediate diagnosis with a view to early and immediate care and treatment wherever possible. This means that, to my mind, the schools, the courts for adults, both general and special, the juvenile courts, the industrial accident boards, the immigration bureaus, the out-patient and dispensary service, of municipal and largely endowed hospitals should all be supplied with ready access to skillful psychiatric diagnostic service. Upon the social firing line, or at all events immediately back of the trenches in the clearing stations for mental casualties, we require expert psychiatric aid. We there require men of good experience and rapid insight who can roughly grasp the problems as problems of insanity, of psychopathia falling short of insanity, of neuropathic conditions requiring special care and of character defects and peculiarities which only the psychiatrist has much grasp of at the present time. Nor would I myself care to be posted on the zone of advance as physician to a juvenile court or an industrial accident board, or an immigration bureau, or in any other of the agencies above mentioned, without the ability to call upon an expert psychologist, one expert especially in mental tests. Again I should be at a loss if I could not have the services of an expert social worker. You will all recognize that what I say about the psychiatrist in the zone of social advance with his *aides de camp,* the psychologist and social worker, has come true in a number of model situations in the country. Perhaps it has been accepted as a principle of mental hygienic procedure.

However, I am bound to say that although these model situations in the zone of social advance do actually exist, yet the progress to securing more such model situations is slow. One reason for this retardation, I feel, is the idea conceived by the rubber stamp gentry that some kind of central bureau in a district is going in the future to render the firing line psychiatrists, psychologists and social workers unnecessary. Particularly as Director of the Psychopathic Hospital in Boston, I am often asked whether our metropolitan problem in Massachusetts is not on the high road to solution on account of the existence and activities of the Psychopathic Hospital. Why, it is queried, should there be

highly special officers in all these somewhat subordinate agencies when the whole matter can be done better and more rapidly, on the department store principle of passing on from examiner to examiner in the Psychopathic Hospital? Why, indeed? It would be an excellent and comparatively inexpensive plan for communities to adopt, if only the plan could be worked. I have to point out that, except in its out-patient department, which is virtually a dispensary along the same generic lines as other dispensaries, the Psychopathic Hospital is not so much in the zone of advance as in the line of communication in the theatre of mental hygienic operations. It is superfluous and very expensive to pass the majority of cases from juvenile courts, schools and other agencies in the zone of social advance through the psychopathic hospital mechanism. If the regimental surgeons back of the trenches can heal their sick, it is superfluous to send them back to field hospitals and base hospitals.

The psychopathic hospital is a sorting machine for cases that cannot be readily sorted on the social firing line, cases which may not deserve internment forever in hospitals for the insane, which may not deserve internment in prisons, which may not deserve elaborate social supervision or other protracted devices of mental care and treatment. I say the psychopathic hospital is a sorting machine to which radiate all those over-complex cases from the zone of social advance, which cannot be immediately diagnosticated and sent forward or backward as the case may be. For example, if in the juvenile court, or the adult court, the expert medical officer can prove to his authorities that by due process of law an alleged criminal is really insane and committably insane, then as a general thing it would be unwise and uselessly expensive to send this patient to a psychopathic hospital. He should be carried to the rear, to the *zone of the social interior,* so to speak, as fast as is consistent with his proper care. The psychopathic hospital is not a sorting machine for cases that can be sorted on inspection or by the simple devices of clinics in the zone of social advance. If a case is thought to be mental, but there is doubt as to committability or proper treatment, or if a special course of brief treatment under an atmosphere of experts is adjudged to be desirable, then the psychopathic hospital can prove its worth. To the hospital radiate doubtful cases from all the clinics in the zone of social advance. From the psychopathic hospital likewise runs another fanwise set of communications, to the zone of the social interior, where exist the well-known mechanisms of social and mental care and treatment known as hospitals for the insane, sanitaria, reform schools, training schools, schools for the feeble-minded, hospitals for epileptics, hospitals for dipsomaniacs and drug addicts, prisons, boarding and placing-out systems and other schemes of social supervision.

The reason why the psychopathic hospital takes this important place in the modern doctrine of mental hygiene organization is not that it can do the work of the zone of social advance better than the firing

line workers; for it cannot. It is not that the psychopathic hospital can do the work of the hospitals for the insane and prisons and the like better than these older mechanisms for this work. The psychopathic hospital cannot do this work so well as institutions manned by experts in the particular fields commanded by these older permanently organized institutions. The value of the psychopathic hospital experience, so important for every psychiatrist, psychologist and social worker of the present day, is not so much the value of experiences on the firing line, though much of this also is available in the out-patient department, nor is it the ripe value of specialistic knowledge in classical psychiatry, criminology and sociology such as is to be obtained by service in the older institutions of what I have termed the zone of the social interior. The psychopathic hospital officers are not so much snap diagnosticians, that is to say, expert offhand observers on the one hand, nor on the other hand are they experts in every field which they must touch. However, they get a certain maturity of comparative analysis which neither the firing line workers nor the interior workers can get. That is why I should advocate a course of psychopathic hospital experience for every person going into the extensive observational work of the zone of social advance, or the intensive, synthetic work of the zone of the social interior.

From this mere sketch of the situation of the psychopathic hospital, it must be clear to my hearers that, as a director and earnest supporter of psychopathic hospital work, I cannot and do not regard it as a panacea in mental hygiene. Before a district deserves a psychopathic hospital, that is, before it can get the most from such a hospital, the institutions and agencies that come immediately in contact with the mental problems of social life must develop themselves notably. They must, to my mind, equip themselves at the present level of social progress with psychiatrists, social workers and psychologists. The moment that two or three of these outermost social agencies get properly equipped for rapid and accurate observational work, then the need for a psychopathic hospital will become apparent, and its great initial cost and comparatively large maintenance expense will seem small to the community in comparison with the fruits of the work.

Some of you may feel that I am myself guilty of the rubber stamp error by providing such a schema of double radiation into and out of the psychopathic hospital. The schema, however, is an inductive one and not born after the fashion of rubber stamps. It provides for democracy of spirit; neither the officers in the zone of advance nor the psychopathic hospital officers, nor the officers of the specialized permanent institutions in the interior can regard themselves as superior or inferior to each other. For all have their special work of observation, comparison and synthesis, if I may choose three entirely inadequate terms to express the major significance of each species of work. By some such plan of organization for the community (obviously founded rather more

upon metropolitan than upon rural conditions wherein large modifications may be necessary) we can provide for the initiative and elbow room of all our workers. Let me insist that that is the great desideratum—not that all the social workers, psychologists and physicians engaged in social work shall run like sheep after some Teutonically conceived ideal of rubber stamp *Kultur*.

This very day we are witnessing the acquisition of a certain number of signatures to a proposal of, to say the least, doubtful value,—a pacifistic proposal supported by numerous ex-officials of our Conference. Many of the workers to whom I have spoken about this proposal, unofficially circulated by some of our worthy ex-presidents, have not been able to explain to me in clear English exactly what some of the phrases in the pacifistic screed really mean, yet some hundreds of signatures out of the thousands of our attendance may very well be secured, simply because we put so much faith in our ex-presidents that we follow them like sheep the bellwether. Here is a very pretty example of the insidious methods of the autocratic principle. I have no idea that our former chiefs sat down deliberately and conceived a design of entering politics with the conference; a design which, however, has been carried out to the letter and is fraught with much danger to the future of the organization. Here is a proposal that the president be asked to deal, at a particular phase of diplomacy, with peace proposals. Whatever the rights of the matter, the whole question is distinctly arguable; yet no particular argument has been afforded unless the Honorable W. H. Taft this afternoon may seize this opportunity (I fancy that he will be found arguing against any such proposal as that of our worthy leaders of former conferences), nor has there been any opportunity for voting. Asked to sign such a proposal, if I refuse, I am greeted with a look of astonishment that I should be willing to vote against the worthy Mr. So-and-So who has done so much for social organization, or the worthy Mr. So-and-So, a great social chief, who is clearly an altruist, and the like.

My predecessor on the platform, Dr. Paton, asked me to say a word about egotism. Dr. Santayana has of course said the last word about egotism in German philosophy. I can add nothing to the point, and perhaps I have said enough about autocracy as the matter stands. The aims and motives of the Great War are doubtless in a large sense the aims and motives of a huge controversy in mental hygiene,—a controversy between forces of autocracy and the forces of democracy. We may regard the German point of view as one of paranoia if we like, that is, as one of exaggerated and morbid egoism. The Germans themselves are querying whether other nations are not possibly victims of mental disease. One German author has soberly inquired whether the "revenge-psychosis" of the French may not perhaps be termed *Psychopathia Gallica,* whereat another German psychiatrist rejoined that to term the French psychopathic was a little dangerous, because "if the

French were psychopathic we, as Germans, would have to feel charity for them".

I have recently been reading in my colleague, Prof. Haskins' little book on Normandy. Dr. Haskins there brings out a splendid distinction between vertical responsibility and lateral responsibility. He points out that in the old days one baron could rob another baron safely if only the suzerain got his share of the loot. The responsibility was from baron to suzerain possibly through a series of under and over-lords, but the under and over-lords had no lateral responsibility to each other for the maintenance of peace or social ethics. I suspect, though I would hardly be required to prove it in the midst of so many alarms, that one great fault in the socialization process that some of us have adopted from German models is the fault of a trace (or more than a trace) of feudalism; that is to say, of an excess of vertical responsibility as compared with lateral responsibility. The worthy chiefs of former conferences, whom I have used as lay figures for my denunciation of the autocratic principle, will naturally not take personal offense at these remarks. I feel that the different elements of our organization should develop a sense of mutual or lateral responsibility to one another along all practical planes. I feel that the rubber stamp method of social organization has failed, is failing, and is bound to fail. I feel that mental hygiene social organization is a matter which is determined by the configuration of the zone of social advance. I feel that expert workers belong in the zone of advance, that expert workers belong in the line of communication and that expert workers belong in the zone of the interior. I feel that unity of social progress is a unity to be obtained *e pluribus*.

Despite this little ripple of the petition about peace terms, I commend to you the spirit of democracy as against the spirit of autocracy.

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ON THE FOCALITY OF MICROSCOPIC BRAIN LESIONS FOUND IN DEMENTIA PRECOX*

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Abstract

The task of microlocalization.

Work of the topographers.

Problems of a pure functionalism.

Anomalies in dementia precox brains.

Crucial importance of histology of normal-looking brains in dementia precox.

Dissociation of parenchymatous and interstitial lesions in certain cases.

The stratigraphical analysis of the cerebral cortex: supracortex and infracortex.

Many effects of mental dissociation (schizophrenia) probably correlated with supracortical disease.

Correlations of hallucinosis and catatonia with infracortical disease.

Topographical analysis: *a priori* probabilities.

Tendency to lobar and lobular focality of lesions.

Delusions: frontal; catatonic symptoms; parietal; auditory hallucinosis; temporal.

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Exceptional delusions of a fantastic nature probably parietal in origin.

Analysis of four cases.

Summary.

A number of investigators of the mind and its diseases are now pursuing the study of the nervous system along lines roughly summed up in the term microlocalization. But this task must not be conceived narrowly as a task of the histoneurologist or the histoneuropathologist. Microlocalization in the nervous system and especially in the cerebral cortex has threads running to the broadest issues of physiology, embryology, and psychology. Yet no one feels that psychologists, embryologists, physiologists, anatomists or even pathologists in the narrower sense of the term, will solve the problems of microlocalization or even demarcate its problems for solution. When the idea of Flourens that the brain parts were mutually exchangeable in function like so many liver parts, was replaced with the ideas of topographical differentiation which we attach to the work of Hughlings-Jackson and of Hitzig, it was as if the naked eye of observation was replaced with a lens of considerable power. The situation now is that this lens of great power, provided by the work of the embryologists, the physiologists and the clinical anatomists, must be replaced with a lens of still greater power, one that will permit us logically to face problems of microlocalization attaching to physiological complications within the single gyrus. It has become commonplace that the brain is a congeries of organs. It is not so well understood that each gyrus is a wonderland of structures and functions in which rich results are to be expected from hard but fascinating research.

As we look over the history of these developments, we of course find that Bevan Lewis had, in advance of his time, laid down the idea of cell differentiation within the cerebral cortex, clearly distinguishing the large cells of the motor cortex, for example. In much of the work of Meynert and Flechsig, the idea of microlocalization can be discerned. However, even in the embryological considerations of Flechsig, the notion of the gyrus as little more than a functional unit is the prevailing idea. Decades were devoted to the isolation of one neurone after another by ingenious technical

methods and to developing the conception of the nervous system as a mass of embryologically and functionally separate units, expressed for example in the neurone hypothesis of 1891. Listeners to the lectures of Nissl became familiar with his ideas of the structural and functional differentiation of the various cortical parts.

The idea of microlocalization scarcely flourished to the full in the last decade of the nineteenth century, however, since workers were engaged in assimilating the principles of Weigert concerning neuroglia and in trying to find qualitatively the same sort of lesions in the nervous system that they were finding in other organs. In fact, this search nearly culminated in the establishment in the years 1899–1904 of general paresis as a definite example of chronic inflammation of the nervous system. The pathologist was here trying to find, and succeeded in finding, certain lesions scattered in different parts of the nervous system, and lesions of qualitatively the same sort throughout. Nor in the rush of new ideas was the material in general paresis used to the utmost in throwing such light as it might on the problem of microlocalization. Thus, had the world been seriously trying to find out the nature and genesis of mental phenomena and symptoms, it would surely have executed elaborate studies in the distribution of the characteristic paretic exudate, with the idea of correlating these local variations in exudate with processes and symptoms. The issue of such an investigation could only have been positive or negative. As it stands, the situation is doubtful. In fact, it may be confidently doubted whether the paretic exudate itself is at all responsible for many of the characteristic symptoms in paresis, for example, grandiosity, amnesia or even dementia, symptoms which are not found to correspond even roughly with the amounts of exudate discovered in the brains.[1] So far as paresis is concerned, very possibly it was more important to determine it to be syphilitic and to embrace eagerly the opportunities afforded of a Wassermann reaction than to pursue seemingly recondite studies in microlocalization. Meantime the topographical anatomists were laying down the results which the pathologist could at last employ without signal loss of time.

When the interesting and fundamental work of Hammarberg appeared on the histology of feeble-mindedness in 1895, it was plain

that this Swedish investigator had had to spend more than half his time, not on the histopathology of feeble-mindedness, but upon normal histology. The situation is now greatly improved with the work of Elliot Smith who showed that even the naked eye will, under proper conditions, detect striking differences between the gyri of different parts of the brain on section, and especially with the work of Campbell who, after an extensive study of the histology of the different gyri, has offered a valuable atlas with a store of functional comments and speculations.

Even before Campbell, Bolton had laid down exact lines concerning the histology of different parts of the occipital region, and had contributed the idea of visuosensory and visuopsychic regions therein. Similar work has been done by Brodmann in Germany, who, however, feels that functional speculations are hardly in point at the present time. Visitors in the private laboratory of Vogt in Berlin some years since must have been impressed with the tremendous elaboration of structures which could be demonstrated in the different parts of the brain, even by so simple a technical method as the method of Weigert for myelin sheaths. For decades also the world has been watching the development of the ideas of Ramón y Cajal, whose labors stand not second to those of Bolton, Campbell, Brodmann, and the rest of the topographers.

In fact, the research situation is theoretically so promising, in view of the progress along all these lines by workers in different countries, that it is a little surprising that so little neuropathological application has been made of the results attained. There seems to be a curious lack of tempo in neuropathological research. Bevan Lewis's ideas of cortex differentiation came, it would seem, too early to affect the main current of investigation. The work of Donaldson in 1889–90 upon the brain of Laura Bridgman has not been followed up by any considerable series of similar investigations, and the Laura Bridgman work apparently led its author to deeper considerations of growth and development rather than to progress along the line of neuropathology.

Again, throughout the nineteenth century there prevailed the Wundtian psychology. The reader of Wundt's analysis of aphasia, for example, is convinced that, despite the profound distinctions

made, the philosopher was no longer a physiologist and set himself the task of antilocalization in the spirit of a system-maker rather than that of an independent seeker after facts and independent interpretations. The idea of the unity of the mind, a very fine-grained and closely interwoven unity, has prevailed in many quarters, perhaps in all quarters, dominating research. To these psychic unitarians it would have seemed almost a pity if anyone could show, for example, an identity of speech processes with processes of the left inferior frontal gyrus. To the conservative wing of the psychic unitarians was added the force of those strictly scientific persons who feel that a fact is a fact and a speculation is necessarily a bad speculation. Thus, for example, if the simpler ideas of Broca concerning speech localization ought to be modified by the modern concepts of Marie, then this is scored as a victory by those holding to the adamantine unity of the mind on the one hand and by those who are sceptical of all scientific results in the psychic field of whatever sort.

Added to this situation was the Freudian tendency. Disregarding the truth or falsity of the Freudian contentions, it is clear that his followers are engaged much more in finding satisfactory categories of a logical nature in which to place globar tendencies shown by their patients than they are in conceiving the brain functional lines along which an hysterical dissociation might proceed, a repression be mechanized, or a censor be enthroned.

Inasmuch as Descartes made a laughable error in assigning the seat of the soul to the pineal body, it is best for the modern to "play safe" by failing to notice both the pineal body and the brain itself and to solve the problem either by denying that it exists or by failing to consider it at all. One of these modern worthies assured me some years ago that there was no doubt a correlation between mental processes and brain processes and that, theoretically, the "needle" of demonstrable lesions could be found in the "haystack" of neuronic brain systems, but that for his part he regarded that problem as "theoretically" solved and desired to get on with something genuine and practical.

Accordingly, the academic psychologists, if we may so term the Wundtians, and the apparently more radical Freudians, have alike

little interest in the matter of microlocalization, that is, in the correlation of mental with brain processes, either normal or pathological.

It is therefore almost with a feeling of being alone in the wilderness that one endeavors to work in the direction of neuropathological correlation: either there is no problem or it is theoretically settled beforehand. A psychiatrist of light and leading asked me one day what, after all, I was about in working along structural neuropathological lines. Anybody with a logical turn of mind and any capacity for observation ought rather to be busily applying the modern categories of psychopathology to clinical psychiatric and neurological material.

If such doubt of the fundamental value of this kind of work could prevail in the mind of a leading psychiatrist, I felt that I could do no better than bring these general ideas before the Association of American Physicians.

It is as possibly throwing light on the problems of microlocalization that the material in dementia precox struck me as of value. To be sure the world was full of a rather empty discussion as to whether dementia precox was an organic or structural disease. Anyone who professes to find brain changes in dementia precox was perforce regarded as a controversialist, engaged in demolishing the pet ideas of some workers who wanted to show that dementia precox was a disease of maladaptation or a disease with so-called Freudian mechanisms. Let me say therefore that to embark on studies of the brain in dementia precox and to find brain changes therein is not at all to deny that dementia precox is an instance of maladaptation of the individual to his environment and not at all to deny the possible importance of Freudian mechanisms in the disease.[2]

In 1906 I began to study the brain problems of dementia precox and collected first-fruits of that study at the Medical Congresses in 1910[3] and 1913.[4] I then showed that the brains of dementia precox subjects were extremely likely to show evidences of anomaly, that even when there was no anomaly, there were very likely to be microscopic changes. To be specific, I found 23 out of 25 carefully studied brains to be significantly anomalous. I do not mean that the anomalies shown were visible the whole length of the demon-

stration hall, as they are apt to be in brains of the feeble-minded, but I mean that the asymmetries and irregularities were of a demonstrable and photographable nature. Friendly critics offered the suggestion that similar anomalies were to be found in brains of normal persons. Unluckily the brains of normal persons, in large numbers, are not available so far as I know in any laboratory in the world, at least in such a state as to permit proper photographic analysis. However, I think I am able to answer these critics by showing that the brains of subjects of the so-called manic depressive psychosis show such anomalies in very few instances.[5] Using certain criteria, I found four-fifths of my dementia precox brains to show such anomalies and but one-fifth of the manic depressive brains were similarly affected.

But it is one thing for a brain to be anomalous and another for it to be functionally disordered. The underlying hypothesis here is that the anomalous regions of the brain are in some sense weak places therein, such that disease of a toxic or metabolic nature, *e. g.*, at puberty, may unfavorably affect the anomalous and poorly constructed region. These considerations I presented with some histological confirmation at the 1914 meeting of the Association in a paper "On the Direction of Research as to the Analysis of Cortical Stigmata and Focal Lesions in Certain Psychoses."[6] Further reflection on this finding led me to the considerations about tissue decomplication developed at the 1915 meeting.

But it was clear that the few cases of normal-looking brains in dementia precox formed the crux of the situation, since the critics of material from anomalous brains might properly charge the investigator with inability to cull out the acquired from the inborn lesions. I therefore determined to study with sufficient intensiveness the brains of cases of dementia precox that showed no anomalies or scleroses. In connection with this study, five brains were investigated, four of them looking in the general direction of dementia precox and one of them in that of manic depressive psychosis. These five brains were the residuum of a series of 120 coming from psychoses of all sorts.

Before I could bring the topographical problem into clear relief I found that in the analysis of these five brains the problem of

parenchymatous *versus* interstitial lesions stood out. At the 1916 meeting I presented the conception that in the nervous system there should be a dissociation of lesions somewhat similar to that in the kidney.[7] To be sure the parenchymatous and interstitial lesions of the kidney are, as is well known, often if not almost always commingled. But the fact that the lesions in these two types of renal tissue are so commingled in a given case militates not at all against the idea that there are, roughly speaking, two kinds of nephritis—parenchymatous and interstitial. The same theoretical situation holds in the nervous system. I found that parenchymatous and interstitial lesions could be dissociated and combined in the nervous system, much as similar lesions could be dissociated and combined in the kidney. To be sure the parenchyma of the nervous system was a parenchyma composed of neurones and their adnexa and the interstitial tissue of the nervous system was complicated by the fact that a great deal of it is neuroglia tissue, embryologically different from the interstitial tissue of the kidney. Nevertheless, it is well known that many of the properties of neuroglia of ectodermic origin are similar to the properties of mesodermic interstitial tissue. The alterations of the mesodermic interstitial tissue also existent in the nervous system are of far less consequence than those of the neuroglia tissue. The one case of manic depressive psychosis, intensively examined, failed to show convincing degrees of parenchymatous lesion, but the dementia precox cases had well-marked parenchymatous disorder, that is, varying degrees of cell loss, to which the interstitial reaction, that is, gliosis, was found to be not at all proportionate. The result of this study seemed to be that parenchymatous disorder, *e. g.*, cell loss and interstitial disorder, *e. g.*, gliosis, must be investigated separately if we are to make headway in this group of cases. Thus it would be decidedly unwise to argue from the data of neuroglia preparations that the gliosis of such and such an area was an indicator of parenchymatous loss. To be sure the gliosis is probably an indicator of something of general or local significance in the brain. But no facile application of the principle suggested by Weigert's neuroglia work could safely be made. Even if gliosis, as a rule, signifies some degree of wear and tear on the part of the parenchyma of the part affected, yet there was no

good evidence that the gliosis was at all proportionate to the parenchymatous wear and tear.

Having thus shown that the brain conditions roughly corresponded to, *e. g.*, renal conditions and that the histopathological analysis of injured brain tissues must look in both these directions in search of successful correlations, I proceeded to a stratigraphical analysis of the finer cortex changes in these brains, presenting results at a meeting of the American Neurological Association.[8] I tried to study separately the nerve-cell losses on the one hand, and the gliosis (including satellitosis) on the other hand, and then to learn whether the character of these changes in the upper and lower layers of the cortex was of any functional significance. For convenience, I wish to term the upper layers of the cortex supracortical and the lower layers infracortical. In fact, it might be well at times to speak of the supracortex and the infracortex. The comparative anatomists seem to have demonstrated the importance of some such distinction and the literature has for some time contained references to the so-called suprastellate (supragranular) and infrastellate (infragranular) layers of the cortex. Omitting controversial details, it appears that almost all workers are committed to the idea that the majority of the supracortical structures are of more recent evolution than the majority of the infracortical structures. As many of the mental functions in man are of recent evolutionary origin and as all signs point to the cerebral cortex as somehow engaged in these processes, it seems natural to assume that the supracortical layers or what we may term the supracortex is the basis of many of these higher psychic functions. With the same plausibility, we may argue that the infracortical layers or what we may term the infracortex is the basis of such mental or quasimental functions as are found in those animals possessing an infracortex and not yet a supracortex.

All these interpretations are independent of any topographical conceptions and for the purposes of generalization one neglects for the moment the important distinction of the archipallium and neopallium, developed by Elliot Smith.

Taking the whole cortex, by and large, modern work seems to me to invest the supracortex with higher psychic functions than the infracortex.

Now in a disease like dementia precox as psychologically formulated by Bleuler, we find the main feature to lodge in what Bleuler terms schizophrenia. This most valuable term expresses what one might call "split-mindedness" or mental dissociation. Much of modern psychology, whatever may be said to the contrary, is founded on the work of the associationist school of, *e. g.*, John Stuart Mill. We should hardly get on without some of Mill's so-called mental chemistry. Well, for better or worse, associative processes must have their obverse in dissociative processes, and the measure of mental dissociation is the degree of schizophrenia in Bleuler's sense. Kraepelin himself who, on purely clinical grounds of a combination of certain symptoms in certain ways, laid down the conception of dementia precox, has so far admitted the validity of Bleuler's conception of schizophrenia as to use the term schizophrenic very frequently in his description of patients. In one of the most exquisite examples of schizophrenia, viz., the finely divided cleavage of speech known as *Wortverwirrtheit*, Kraepelin has constructed the term *schizophasia* on the analogue of schizophrenia.

We mean then by schizophrenia a process of mental dissociation, a psycholytic or ideolytic process. The cleavage may lie between the intellect and the emotions, such that the emotions do not at all fit the ideas entertained by the patient. Or the cleavage may lie between the emotions and the will, and a bizarre-looking conduct may fit a certain quasinormal state of feeling. But the cleavage is not necessarily so elective and total. The cleavage may be within some particular train of thought. Here is schizophrenia in its best display. Instead of the regular train of thought, we may have a train of thought like a telegraph despatch, or even a train of thought suggesting in its obviously inappropriate sequence a cipher or a set of types reduced to "pi." But absolutely "pied" thinking is not the rule and the so-called word salads are often but passing phases in the development of a dementia precox course.

Here is not the place to expound or to describe schizophrenia. Inasmuch, however, as many of the schizophrenic effects that strike the observer as so bizarre in dementia precox thinking are probably correlated with the operations of the outer layers, that is, of the supracortex itself, you have to inquire whether cases of this order

show lesions preferably in the supracortex. Of my 4 cases of dementia precox, all showed lesions in the supracortex in the nature of cell loss and of more or less disproportionate gliosis, except 1 case. This case was one approximating the so-called paranoia, a disease perhaps allied to dementia precox, but often running years or throughout life without any of the bizarre psycholytic, schizophrenic, thought-splitting phenomena here under discussion. For paranoiacs of this rare description (there may be in a dozen institutions in Massachusetts at this time not more than 25 or 30 of these particular cases) present quasinormal appearances that have been described sometimes as psychic malformations and often give the impression of ability to fit into a reconstructed world with the patients in the center as Kaisers or I. W. W. chiefs therein. In short, many of these paranoiacs, victims of the severest and most intractable psychosis known, are nevertheless without a trace of mental splitting such as I have hinted at above. And in fact the paranoiac in question showed no signs of mental dissociation, but rather showed signs of what might be termed a hypersynthesis of such facts in her environment as fitted her morbid ego and her jealousies. What I am getting at is that the psychopathological analysis of this case of paranoia precisely did not demand any evidence of supracortical disease; in fact, had there been supracortical disease of any moment, we should have wondered whether there had not been some disintegration of the patient's personality, at any rate at the close of her life. It is interesting that this particular paranoiac did show some infrastellate disease which, as will be mentioned below, I regard as correlated with the auditory hallucinations which she showed.

The other 3 cases intensively examined (I may recall that notes were made as to different types of cell loss, gliosis, satellitosis, vascular changes, etc., for each layer in each of twenty-five to thirty areas of the cortex in each subject) showed signs of supracortical disorder as well as of infracortical disorder of varying degrees in different parts of the cortex, and all 3 of these cases showed signs of schizophrenia. This evidence is at least suggestive that supracortical disorder is necessary in schizophrenia. I felt satisfied in my own mind, from the results of the work, that not only is the

cerebral cortex the proper study of the psychologist and the psychiatrist, but that the supracortex is in a still more intimate sense the proper study of the psychologist or psychiatrist who has to deal with the higher functions of what may be called psychic association or synthesis. To my mind, such studies, if in future confirmed, will throw an important added light upon the problems already partly illumined by comparative anatomy.

Let me insist here, if it is not superfluous, upon the fact that I am not proposing an hypothesis to the effect that every time a mental split occurs, a cell drops out of the supracortex. I do not even see in my mind's eye a dendrite dropping off a supracortical cell to signalize the cleavage of an idea. There is very properly much fun to be made of workers who regard ideas as fine transparent substances poured in and out of nerve cells as wine in and out of goblets. Aside from the problem of parallelism and interactionism suggested by the figure that regards brain cells as vehicles for ideas, my own contentions deal rather with the observational facts. I would regard all gliosis as merely indicating and not defining the type of cortical disease in question and I should regard the nerve cell losses or other changes as likewise nothing but indicators of the kind of thing going forward, or perhaps as indicators that something or other is going forward in the injured tissue. To be sure, I would think that destructive brain changes would be more likely to be associated with mental dissociation (with schizophrenia, gliosis) than with other types of synthetic or redistributive mental processes. Supracortical neurone destruction, to put the whole matter in a formula, is to my mind correlated with schizophrenic processes. What, you may well ask, is to be correlated with hypersynthetic, falsely synthetic or redistributive mental processes of the mind twist sort? For the present I, for one, have no idea what cortical process corresponds to these non-destructive, morbid, psychic processes. Mind lack and mind loss *should* correspond with cell lack and cell loss. What we term figuratively mind twist, *e. g.*, false reasoning without loss of sensory intake and power, without loss of memory images, without loss of power of expression, without any evidence of coarse loss of function, remains as a process not readily stateable. Perhaps we must fall back here upon

the idea of physicochemical changes and of anomalous distributions of energy in normal neuronic systems. But these are speculations so remote from observational facts that so far as I can see they cannot even do the observer any harm.

Besides schizophrenia there are other bizarre effects in dementia precox. Perhaps the strangest thing in the world is catalepsy. Catalepsy, catatonia, cerea flexibilitas are not infrequent symptoms in many cases of dementia precox. They all have the character of muscular hypertension somewhat suggestive of hypnotic effects or again of the effects of drugs upon the muscular system. Sometimes the phenomenon of negativism and resistivism reminds one of the Sherringtonian experimental effects showing innervation of antagonistic muscle groups. Possibly these processes are to be explained upon some general line as schizophrenia; possibly they are due to some kind of lysis; some workers are inclined to use the concept of inhibition very freely in the attempt to explain them. They are often a transient phase recurring again after comparatively normal intervals in a schizophrenic patient, and it seems impossible to evoke them by any form of stimulation of the patient.

In a general way, such conditions as catalepsy seem a good deal more like phenomena producible in lower animals than would schizophrenia. Some of the hypnotic or pseudohypnotic appearances in lower animals roughly resemble the catatonic effect. It was in a case of catatonia that Alzheimer first stated that there were nerve-cell changes in dementia precox and it will be remembered that he placed these changes (chiefly gliosis) in the lower layers of the cortex. To a certain extent, my own investigations here confirm those of Alzheimer; at all events, in the cases showing catatonia there were lesions in the infracortex.

Another symptom not at all so bizarre as schizophrenia and catatonia is hallucinosis. Hallucinations are rather quasinormal effects that the normal person rather readily understands and that occur in a great number of mental diseases. Hallucinosis is in short a far less pathognomonic symptom for dementia precox than is either schizophrenia or catatonia. Many forms of hallucination seem to be of so simple a nature that they could well be elicited in the lower animals could we only get at the psychic interiors of

the lower animals to learn their mental contents. *A priori,* therefore, it seemed to me that the disease process underlying hallucinosis ought rather to be in the infracortex than in the supracortex and there was some confirmation of this idea in the cases studied. I mentioned above the fact that late in the disease the paranoiac patient developed auditory hallucinosis. She showed infracortical cell loss of a mild but distinct degree. It will be remembered that the brain of this case was the most nearly normal of all the brains so far studied in the whole series.

The stratigraphical analysis of these brains accordingly proved very alluring. I seemed to get evidence that the supracortex was a region whose disease might well spell schizophrenia, whereas the infracortex was a region whose disease was related with lower level symptoms, such as catatonia and hallucinosis. The supracortex and the infracortex would then be regions of differential interest for the psychiatric investigator, who would seek in the supracortical region for evidences of higher intellectual disorder in the field of association, combination, and abstraction, and in the infracortex for evidences of lower forms of mental disorder, such as catalepsy and hallucinations. I would not stick on the question of higher and lower functions at this point. Catatonia may well be a far more complex matter than many forms of intellectual dissociation; I call it lower because it seems to me that a simpler organism could show catalepsy than disorders of combination and abstraction in the intellectual field.

I have recently reviewed the cell findings in the different cortical areas, assuming that I should find rather suggestive topical correlations between special kinds of symptom and special loci. I assumed that *a priori* no one would be likely to hunt for the source of auditory hallucinations in the smell zone as defined by the comparative anatomists and that one would hardly be likely to look for the basis of visual hallucinations in the superior temporal gyrus!

Perhaps in accordance with the opinion of the learned psychopathologist above mentioned, it is *a priori* certain that in point of fact auditory hallucinations have their basis somewhere in the temporal lobe and visual hallucinations somewhere in the occipital region, and why undertake a superfluous task of setting out exactly

where and under what conditions they occur? We ought to be greatly helped in this matter by the focality of lesions in dementia precox.

In the beginning I had been attracted by the fact that there was often a lobar or lobular hypoplasia, atrophy or sclerosis in these brains. In fact I had tried, reasoning from the gross data alone, to draw tentative conclusions as to the main lines of functional differentiation in the symptoms of dementia precox.

Noting how difficult was the distinction between inborn anomaly and acquired lesion and how uncertain one might be that a given hypoplasia had anything to do with a given disorder of function, I felt that I ought perhaps to investigate cases without gross lesions.

Now one of the most generally significant results of this intensive examination of numerous areas and all layers in dementia precox brains is that there is a certain focality in the microscopic lesions. I still find the focality to be rather a lobar or lobular one than an intragyral focality. To be sure my colleague, Dr. H. I. Gosline, has made some interesting observations upon some exquisitely focal lesions of intragyral distribution, possibly related with tuberculosis, in certain dementia precox brains.[9] Gosline has endeavored to show that the situation of these lesions (demonstrable by fat stains) is such as to fit with my own older contentions as to the relation of certain symptoms to particular regions. Notably Gosline endeavors to confirm my idea of the correlation of catatonia to postcentral lesions. My own work, as here reported, deals not with results of fat stains but with the results of the study of cell losses by the ordinary tinctorial methods for determination of tubercle, at least with nerve cells (Cresyl violet after formalin fixation). I have tried to show the cell losses thus made out as a little more reliable than the fat-stained cells and deposits that Alzheimer, Cotton and Gosline have used. Using the methods of these observers, I obtain too rich a display of lesions, as a rule, to permit correlation.

The kind of focality of lesions which I believe to have demonstrated in these brains is not intragyral, but lobar and lobular, or in certain cases a focality of lesions affecting the whole gyrus. By and large in these brains, as one examines the different layers

within a gyrus, the layers are apt to show throughout the gyrus the same kind of lesions, whether gliosis or cell losses. In short the focality of these lesions is not the focality of tubercles, tumor masses, glanders lesions or exudative lesions. It is rather a focality such that, *e. g.*, the postcentral gyrus on one side is affected in a certain way throughout the gyrus with such and such layers homogeneously affected. The postcentral gyrus of the other side is relatively normal.

What now are the functional results of a study of the topographical distribution of the lesions just mentioned? Can we by focalizing attention on certain gyri discover functional correlations of lesions with certain symptoms?

The functional correlations of my dementia precox studies published in 1910 and 1914–15 were summed up from the topographical point of view as follows:

1. Delusions are, as a rule, based on frontal lobe disease.
2. Catatonic symptoms are, as a rule, based on parietal lobe disease.
3. Auditory hallucinosis is, as a rule, based on temporal lobe disease.

Re the frontal lobe correlation with delusions, I found an exceptional group of delusional cases without frontal emphasis of lesions; but I found a plausible reason for these exceptions. Upon analysis, the non-frontal cases of delusion formation turned out to be cases with a tendency to what may be termed hyperphantasia, that is, to an elaboration of fantasies not more than half-believed, or at any rate not fully believed, by the patient. It is clear that from what we know of the probabilities of localization in the brain, both from a comparative and anatomoclinical point of view, the parietal lobes might plausibly be implicated in the function of imagination and that parietal disease might well be attended with functional disorders of imagination, such as overimagination or what is here termed hyperphantasia.

It is plausible to argue that because the parietal tissue, newly evolved in the higher animals, lying between the tactile postcentral region and the visual occipital region and the auditory temporal region, should be a tissue related with apparatus for combining

these sensory data, a tissue which might unobjectionably be termed a center for combining percepts and concepts involving two or more forms of sensation or perception. Whatever the merits of this *a priori* contention, at all events I found a tempting correlation of fantastic delusions with parietal lobe atrophy.

I summed up the anatomical situation as follows:

"On the whole, the correlation between delusions and focal brain atrophy (or aplasia capped by atrophy?) is very strong, particularly if we distinguish (1) the more frequent form of delusions with frontal-lobe correlations from (2) a less frequent form with parietal-lobe correlations.

"The non-frontal group of delusion-formations, the writer wishes to group provisionally under the term *hyperphantasia*, emphasizing the overimagination or perverted imagination of these cases, the frequent lack of any appropriate conduct disorder in the patients harboring such delusions, and the *a priori* likelihood that these cases should turn out to have posterior-association-center disease rather than disease of the anterior association center. This anatomical correlation is in fact the one observed."

My previous work had also suggested a possible correlation between catatonic phenomena and parietal (including postcentral) disease. Ten of 14 definitely catatonic cases yielded gross lesions in the parietal or other post-Rolandic regions. Of the remaining 4, 2, negative in the gross, proved to be microscopically altered in the parietal region, and there were indications of a correlation in the remaining 2 cases. It must be remembered with respect to correlation that I am using the term "catatonic" here as the name of a symptom. I am not offering a correlation between anatomical or microscopic lesions and the catatonic form of dementia precox.

For example, the symptom *cerea flexibilitas* is not necessarily an indicator that the victim belongs in the so-called catatonic form of dementia precox, despite the fact that *cerea flexibilitas* is one of the prettiest examples of catatonia ever described. Four of the 5 cases in the 1914–15 series that showed *cerea flexibilitas* had parietal anomalies or atrophies and the fifth case, though it was entirely negative in the gross, was a case which showed an extreme

degree of satellitosis in the postcentral region examined microscopically. These observations and certain *a priori* considerations led me to formulate the idea that catatonia, and particularly *cerea flexibilitas* was a form of disorder of kinesthesia.

Re the correlation of auditory hallucinosis with temporal-lobe lesions, it is easy to see that such a correlation ought to exist if any gross correlations at all were expected. In point of fact, 9 of my 12 hallucinating cases have had temporal lobe atrophy or aplasia. One of the 3 remaining cases had ample microscopic changes in the temporal lobes, 1 was clinically somewhat doubtful and, in fine, only 1 of the 12 hallucinating cases could be safely said to have neither gross lesions nor important microscopic lesions in the temporal area.

To sum up then, (1) we expect frontal-lobe lesions in cases of delusion formation, provided that the delusion formation is not of the fantastic and overimaginative sort, whereupon parietal lesions would be expected; (2) we expect parietal lesions associated with catatonic symptoms; and (3) we expect temporal lobe lesions in cases of auditory hallucinosis.

Accordingly, I have collected the microscopic data in the 4 cases at present under discussion under these heads—frontal, parietal and temporal. For convenience I have divided the cortex into three portions—frontal, parieto-occipital and temporosphenoidal. The following table indicates the extent of nerve-cell loss and of neuroglia proliferation, separately in the three regions mentioned:

	I (10.9) 14 years.	II (12.41) 10 years.	III (12.47) 2 years.	IV (11.36) 20 months.
Frontal loci	9	9	9	6
Nerve-cell losses . . .	5	44	35	34
Neuroglia proliferation .	7	8	4	3
Parieto-occipital loci . .	10	9	10	7
Nerve-cell losses . . .	17	47	54	19
Neuroglia proliferation .	8	3	2	8
Temporosphenoidal loci .	10	10	10	6
Nerve-cell losses . . .	9	48	50	25
Neuroglia proliferation .	5	9	1	0

It is clear from inspection of the table that Case I (10.9) stands out as a case in which the parenchymal disease is especially parieto-occipital, since in ten loci, seventeen instances of nerve-cell losses in different layers were found. Here then should be a case with

catatonia unless perchance the lesions in the parieto-occipital area should be exquisitely occipital and not affect kinesthetic areas. In point of fact the lesions enumerated are all in the postcentral, superior and inferior parietal areas and are especially marked in the inferior part of the postcentral gyrus and in the superior parietal region on both sides.

This patient was one that developed characteristic catatonic symptoms, including *cerea flexibilitas* and attitudinizing in the last two years of her life. She also had auditory hallucinosis in those years and showed, as the table indicates, a number of parenchymal losses in the proper area, especially the superior temporal gyrus. Both the parietal and the temporal lesions were largely infracortical in distribution, in accord with the argument mentioned above as to the infracortical origin of catatonia and hallucinosis. It will be noticed from the table that there were comparatively few lesions in the frontal region. The case was one of delusions of jealousy and might have been expected to have shown frontal lesions which, however, were not greatly in evidence. However, upon closer analysis of the delusions, it will be found that they are of a synthetic character and not of a schizophrenic character. They are quasi-normal, and it is not easy to conceive just what histological basis such delusions *ought* to have. At all events none of any consequence were found and the frontal region remains practically destitute of any parenchymal loss and has exceedingly slight evidence of gliosis. Accordingly we may suppose that we are getting histological evidence in this case merely of the terminal hallucinosis and catatonia and not of the essential paranoia of the patient.

If we turn to Case II (12.41) we find a totally different situation. The amount of parenchymal loss, as indicated by the number of loci, is far larger than in Case I (10.9). In fact there are four times as many loci affected, and the lesions are scattered throughout all three regions—frontal, parietal, and temporal. There are relatively more parenchymal losses in the frontal region than in the temporal region and relatively more in the temporal region than in the parietal region.

If we took these data on their face value, we should say that this case ought to be predominantly delusional but also hallucinatory and catatonic.

There was some question whether the patient was not slightly feeble-minded to start with. However, her command of theological verbiage was large and she was always self-supporting up to the time of her psychosis. My point in mentioning this query is that very possibly some of the apparent parenchymal losses are actually due to an initial lack of cells. The interpretation of an initial *lack* of cells in any of these areas would of course look in a different direction from that of an initial *loss* of cells. An initial lack in frontal cells might not at all suggest delusional tendencies; an initial lack of superior temporal cells likewise might have nothing to do with auditory hallucinosis. And a simply constructed parietal mechanism would not necessarily entail disorders of kinesthesia with the production of catatonia.

But taking the parenchymal deficiency as signifying initial *loss*, let us inquire what the symptoms of this case are. Was it predominantly delusional? She showed hallucinations of a doubtful but probably of a hypnagogic visual nature. It is not clear that she ever had auditory hallucinations. The appearance of parenchymal loss has proceeded in all these areas to a marked degree, so marked, in fact, that it is rather surprising that there was no gross evidence of brain atrophy (the brain weighed 1130 gm., possibly a slight reduction from the normal weight; the patient was cardiac and there was such a condition of infiltration of the brain tissue that we can *post hoc* readily believe the brain was slightly and diffusely swollen). In short, the lesions in this brain are so numerous and widespread and so affect both the supracortical and the infracortical regions that correlations of the nature here attempted are impossible. One can find a correlation between almost any symptoms and some properly situated lesion in this case. The patient was deluded, hallucinated and catatonic and had frontal, temporal and parietal lesions.

Let us for the sake of argument, regard the frontal lobe delusion correlation as established; let us regard the parieto-occipital correlation with "feelings of being hypnotized" and the "black cat jumping over a pitcher" as indicators of disease in the parieto-occipital or occipital areas. How then shall we interpret the absence of auditory hallucinations? Without invoking the poverty of

clinical observation and the fact that the patient may have kept auditory hallucinations to herself (she was in point of fact often engaged in vituperative language, some of which may have been of hallucinatory origin or of a responsive nature), we must be interested in the fact that some of the temporal lesions shown are exceedingly heavy and affect both the supracortex and the infracortex. Suppose infracortical, temporal-lobe lesions of a nature suitable to produce hallucinations (we are still ignorant just what sort of lesion ought to produce hallucinations), would it be too speculative to suggest that the existence of severe supracortical lesions might well interfere with the proper combination and abstraction of the infracortical data in such wise as to produce conscious hallucinations? That is to say, the total supra- and infracortical mechanism may be conceived to be so altered that though the conditions for hallucinosis are present in the infracortical zone, the hallucinations are not purveyed through lack of finer connections with the higher psychic apparatus mediated by the supracortical layers. Naturally I do not wish to claim more than speculative value for such considerations. It is along these lines, however, that I feel that much important correlation will be secured in future.

If we inspect the table of local distribution of lesions in Case III (12.47), we find conditions approximating those of Case II (12.41), although the duration of Case III is far less than that of Case II. We find here that the parieto-occipital region has numerous lesions and slightly in excess of lesions, also numerous, in the frontal and temporal areas. The case was a catatonic one, and in fact showed both *cerea flexibilitas* and catatonic stupor and would probably be classified as a case of catatonic dementia precox. It appeared that she had auditory hallucinations which she tried to keep out by covering her head with her arms; but the content of these hallucinations was never made out. An attitude of apprehensiveness, an early phase in which she constantly repeated the words "Father Patterson," "Father Patterson," led us to the idea that she had delusions of persecution or else that she was extremely apprehensive and threw a delusional covering over every-day features of the environment. The most pronounced regions of cell loss were in the infracortical portions of the parietal and temporal regions.

When we come to the analysis of Case IV (11.36) we find that the parieto-occipital lesions are far fewer in number and that the case is more one of losses in the frontal and temporosphenoidal areas. This case, therefore, should show delusions and possibly auditory hallucinations. She never showed convincing catatonic symptoms, although she showed a marked mania and hyperkinesis somewhat suggestive of manic-depressive psychosis. She showed characteristic auditory hallucinosis, and it appears that most of her antisocial acts of a mischievous and silly nature were based upon these hallucinations. That she showed definite delusions, *e. g.*, of persecution or of poisoning, is doubtful. We know little of her history immediately after the onset of mental symptoms which followed confinement in a hospital for consumptives. Throughout here period of observation in a hospital for the insane she showed elation, erotism, mischievousness and silliness somewhat suggestive of manic-depressive psychosis and again somewhat suggestive of the elation of certain paretics. The cell losses in the frontal region were numerous and both supracortical and infracortical in distribution. It is interesting that of all four cases, this one was the case which showed a marked myelin-sheath disorder, a disorder so marked as somewhat to suggest the disappearance of fibers in paresis. The supraradiary fibers were absent in all the frontal regions examined and there was a thinning of fibers in the white matter underneath. In short, the disorder in this case was much more than a fine disorder of minor cell mechanisms and approached the more general and globar disorder of paresis, though of course without exudation. Upon some such lines I would be tempted to try to explain the absence of definite delusions except those based upon hallucinations and irritability. I should be tempted to regard the frontal lobe lesions as of such degree that we might think of the case as one that had lost its frontal inhibitions. At the same time, it must be remembered that no coarse atrophy of the brain had supervened, unless we regard the brain weight—1140 grams—as demonstrating brain atrophy.

Summary. Thanks to the work of Elliot Smith, Bolton, Campbell, Brodmann, Ramón y Cajal and others, the neuropathologist can now afford to attempt finer functional histological correlations.

in the field of mental diseases, thus aiding in the problems of microlocalization. The antilocalizing tendencies of the Wundtians and the interest in merely logical categories taken by Freudians should not interfere with progress in microlocalization. Dementia precox, for example, can be called a matter of maladaptation of the patient to his environment or of the patient to himself and also a disease characterized by cortical changes.

Previous work had shown anomalies in a high proportion of dementia precox brains and in a correspondingly low proportion of the brains of manic-depressive subjects. These anomalies may well be interpreted as weak places in these dementia precox brains, and the brains in fact are apt to show scleroses and atrophic processes over and above the anomalies. But certain perfectly normal-looking brains in dementia precox also show the same microscopic changes in lesser degrees than are found in the anomalous sclerotic and atrophic brains. The problem of the present communication has been to work out the focality of these microscopic lesions in a few normal-looking brains studied with unusual intensiveness. In the same series of brains, work of previous seasons had shown a dissociation of parenchymatous (neuronic) and interstitial (neurogliar) changes, indicating a tendency on the part of cortex pathology to resemble the pathology of the kidney. But the majority of brains show mixtures of the parenchymatous (neuronic) and the interstitial (neurogliar) lesions. Recent work in comparative anatomy indicates the rather fundamental importance of distinguishing the functions of the upper cortical layers (what may be called the supracortex) from the functions of the lower cortical layers (what may be called the infracortex). The finer processes of mental dissociation (schizophrenia) *ought* to be correlated with lesions of the supracortex, and such lesions were found in cases with evidence of schizophrenia.

On the other hand, in a case of delusions characterized by no splitting (schizophrenia) whatever, but rather by a process of overelaborate synthesis, there was no evidence of supracortical disorder and, in fact, no proposal can be made for any histological correlation with this process of oversynthesis.

Other processes equally characteristic of dementia precox but

logically far simpler in their make-up, such as auditory hallucinosis and muscular hypertension (catatonia) received suggestive correlation with processes in the lower layers of the temporal and parietal regions respectively.

So far as the tissues of these four cases go, there is little or nothing inconsistent in the findings with the hypothesis that ordinary (non-fantastic) delusions are correlated rather with frontal than with otherwise situated lesions; but the supracortical type of delusions found in certain long-standing paranoiacs whose fine mental processes run in a quasinormal manner, find no special correlation in any region, and the probable lines on which this problem is to be solved remain obscure.

So for auditory hallucinosis, the work seems to afford the expected correlation with temporal lesions. In one case, however, temporal lesions of considerable severity were not attended in life by hallucinations of hearing, but in this case there was also a severe supracortical disease of the temporal region, and it may be that for the production of hallucinations, some congress is necessary between the activation of the supracortex and infracortex respectively.

In previous work on this series the brains had indicated a postcentral and superior parietal correlation for catatonia whose muscular hypertension was accordingly regarded as very possibly a kind of morbid kinesthesia. Present work suggests that the anatomical correlation is not merely to the postcentral and parietal regions but still more specifically to the infracortical parts of these regions.

It may be suggested that the lesions found in samples of tissue in the postcentral, superior parietal, inferior parietal and superior temporal regions indicate a certain systemic tendency in the underlying processes. For these lesions were bilateral and occurred, as it were, in two continuous sheets of tissue on both flanks of the brain in one of the best defined of our cases; these flank lesions were not attended by any similar lesions of the frontal, precentral, occipital and lower temporal and smell regions. The nature of a process which could mildly affect nerve cells and neuroglia on two sides of the brain and also specially affect the infracortical rather than the supracortical portions of these affected sheets of tissue remains a mystery. It is perhaps no greater mystery than that which

attends the distribution of lesions in the spinal cord of pernicious anemia. It remains unsettled whether these lesions are secondary in point of time to a non-cell-destructive phase in the disease, or whether the lesions of which these microscopic effects are indicators began *pari passu* with the symptoms—that is, it remains a question whether we are dealing with the excess wear-and-tear process of cell mechanisms morbidly employed or whether the morbidity of neural function is an exact equivalent of the neuronic and neurogliar morbidity.

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LVI

THE GENERA IN CERTAIN GREAT GROUPS OR ORDERS OF MENTAL DISEASE*

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I want to present to the American Neurological Association certain amplifications of material presented in 1917 as a key to the practical grouping of mental diseases. Under the eleven groups of mental diseases defined in 1917, I wish to place such practical subdivisions as seem to me confirmed by American psychiatric experience.

As I find that many persons hardly distinguish between a classification and a key and labor under the impression that I am trying to erect a novel classification of mental diseases, let me insist that I am proposing nothing but a key to the classification of mental diseases according to the entities which I find in common diagnostic usage. I am elsewhere insisting on the extraordinary unanimity which American psychiatrists are now displaying on the matter of psychotic entities. There is, in fact, hardly enough controversy to indicate a healthy progress in the matter of theoretical psychiatry. (There is, to be sure, one large controversy concerning the nature and dimensions of psychogenesis and the part it may play in sundry mental diseases; but this controversy has to do with more general aspects of psychiatry than the question of its contained psychotic entities. Nothing is more hopeless than a discussion, for example, of psychogenesis in dementia praecox when the controversialists do not agree as to the clinical symptomatology of the cases under discussion.) This unanimity of view as to the psychotic entities of modern psychiatric science is so marked that a committee of the American Medico-Psychological Association has been able to formulate an acceptable list of such entities now in process of adoption by most of the institutions for the insane in the country. The progress in mental hygiene secured by this universal adoption of a list of psychotic entities is certainly a subject for congratulation.

PURPOSES OF THE AUTHOR'S INVESTIGATIONS

What I have been attempting of recent years on the basis of the diagnostic sifting-machine material afforded by the Psychopathic Hospital is to study the logical processes of psychiatric diagnosis and to

* Read in abstract at the Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1918.

find, if possible, some simpler ways in which to arrive logically at one or other of the psychotic entities which we virtually all agree on.

I have placed some larger considerations on this matter of the "process-types" of diagnosis in a paper read this year before the Association of American Physicians, to be published in the *Journal of Clinical and Laboratory Medicine.* The paper is entitled *"Diagnosis per Exclusionem in Ordine:* General and Psychiatric Remarks." I do not need to rehearse the points of this paper before the American Neurological Association. I was, in fact, trying to read something of a lesson to the diagnosticians of the eminent internist group represented by the Association of American Physicians, calling to their attention the need for more elaborate logical methods of approach to diagnosis in psychiatry than in many branches of medicine. Some of the elders amongst the internists had for years denounced the method of diagnosis by exclusion; one of them said that the method was bound to fail because of our ignorance of pathology and went on to say that diagnosis by exclusion was a tedious method. Of course, tediousness ought not to stand in the way of accuracy, and pathology is bound to remain imperfect for many decades, not to say centuries. The fact is that in fields of diagnosis where there are no indicator symptoms, the method of diagnosis by exclusion is unconditionally necessary; for, in the absence of an index of differentiation or indicator symptom (or "presenting symptom" as Dr. Richard Cabot sometimes calls it), the diagnostician is bound to take into account all forms of mental diseases when he is trying to eliminate and differentiate the particular psychosis displayed by his patient.

Hence, I went into some detail in the paper mentioned, on a method of diagnosis which I called *diagnosis per exclusionem in ordine.* The central part of the idea had already been presented in the paper entitled "A Key to the Practical Grouping of Mental Diseases," presented before you in 1917.

The advance which I want to make this year is implicit in the method of the key presented last year. Last year I suggested that the tyro in diagnosis might well consider and exclude in sequence the great groups of mental diseases A, B, C, D, etc. I put A before B, B before C, etc., simply because the methods of diagnosis in Group A appealed to me as more certain, practical and general in their scope than the method available for Group B; the same for the methods of Group B as against those for Group C, etc.

This year I want to set down the subgroups of mental diseases which it seems to me practically all of us admit exist (if we admit that any entities whatever exist), in a proper diagnostic order. I want

to extend the principle of orderly diagnosis, that is, of *diagnosis per exclusionem in ordine* (genera) under groups (orders, in the botanical or zoological sense). Now I must acknowledge at the outset that the farther we go into detail, the less unanimity must a priori be expected in the psychiatric world. Accordingly, I would concede that my proposals are bound to be far less acceptable in their details than the proposals in the more general key to the main orders of mental diseases presented in 1917, but if the principle of exclusion in order be accepted for practical diagnosis, then I shall have no quarrel with those who feel that the entities are too many, too few or even non-existent.

IMPORTANCE OF GROUPING DISEASES

One more general remark: I feel that the history of modern developments in logic indicates that the part of order is the one part which has undergone great developments in recent years. We have discovered that though we cannot always measure things, we can sometimes put them in order unmeasured. It seems to me that the development of orderly diagnosis is quite on the carpet for modern workers. It may not be superficial to say that expert diagnosticians may not need to employ the method of diagnosis by exclusion in order simply because the facts in a given case may immediately suggest to them (by processes of mere inspection or of very rapid comparison) the right diagnosis. Time and again, however, the best experts fail in their attempt to apply the methods of diagnosis by inspection and by comparison, and surely the inexpert youthful psychiatrist needs some key to guide him. How frequently in the clinic do we find that the youthful diagnostician is by very little emphasis here and there able to press the phenomena of his case either into the dementia praecox or the manic depressive group or into the senile or the focal brain disease group, respectively. The point of this difficulty lodges in the fact that there are practically no indicator symptoms in mental diseases, and actually any symptom you may specify is quite able to lead you in any one of the main diagnostic directions. Let a young diagnostician of the dogmatic or slightly paranoid type get the initial idea that a case belongs in the dementia praecox group, he will be able to defend his thesis against all comers by the use of symptom lists founded on the very best textbooks; in fact, the better the textbook the easier for the young tyro to carry his point — for the time being.

Following are tabulated suggestions for the generic classification of mental disease groups, each group followed by some general remarks.

I. Syphilopsychoses* (the syphilitic mental diseases):

Genera:

General paresis
Juvenile paresis
Nonparetic forms:
Meningitis
Vascular
Gummatous

Less common genera:
Syphilitic feeblemindedness
Syphilitic epilepsy
Tabetic psychoses
Syphilitic paranoia

Of course, the syphilopsychoses are by no means coterminous with neurosyphilis. The term neurosyphilis generally taken, must be supposed to include both the syphilopsychoses and the syphiloneuroses. The systematist will find a certain difficulty in placing many forms of neurosyphilis amongst the psychoses and the neuroses, respectively. We are here dealing with the psychoses, and our classification does not include the neuroses.

If one were asked how to distinguish the syphilopsychoses from the syphiloneuroses, one would have to reply on practical grounds that, if the case showed psychotic symptoms, it should be placed among the psychoses even if there were also present, is is usually the case, a number of neurotic symptoms. In short, owing to their practical significance, psychoses might be supposed to have the first call in classification as against neuroses. On this account the disease commonly known as general paresis would fall amongst the syphilopsychoses, despite the existence therein of any number of symptoms pointing to nonpsychical part of the nervous system. On the other hand, the disease commonly known as tabes dorsalis would best be placed amongst the organic neuroses, despite the appearance in tabes from time to time of a few mental symptoms. If, however, a case of tabes develops symptoms of a paretic nature, then the common rule is to term the case one of taboparesis. If in the course of the tabes certain characteristic excitements with hallucinations appear, then we have a rare entity known as tabetic psychosis. I am not sure that there has even been a well established case of this disease, tabetic psychosis, in the Psychopathic Hospital clinics amongst 10,000 admissions. So much for the general relation of the syphilopsychoses to the syphiloneuroses.

The issue is a practical one, and decision is made on the appearance of psychotic symptoms in the case. If these dominate the scene, then

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*Re-syphilopsychoses: Dr. Solomon and I in a recent case-book tried to bring order into the nomenclature of neurosyphilis by reducing the main forms thereof to:

(*a*) Paretic
(*b*) Tabetic
(*c*) Diffuse
(*d*) Vascular
(*e*) Gummatous
(*f*) Juvenile

the case should in my opinion be termed syphilopsychotic. Of course, if the syphilitic infection precedes and in a psychogenic way occasions a neurasthenia, then from this point of view we should not be dealing with a case of syphilopsychosis, but with a case of psychoneurosis. If, as in one of the war cases, a syphilitic infection appears to bring about an epilepsy, we are not dealing according to this grouping with an epilepsy which is syphilitic, but an epilepsy presumably brought about in some psychogenic way and only indirectly due to the operations of spirochetes. These two exceptional diseases might be then named psychoneurosis syphilogenica and epilepsia syphilogenica, in which we place in the adjective the exciting factor and place in the abstract noun the general nature of the disease in question.

Syphilopsychoses, then, are diseases in which the psychosis is essentially spirochetal. Where the spirochete acts after the fashion of an occasioning factor, it would seem wiser in the interests of the patient to place the disease elsewhere.

A note on the order in which the genera under the syphilopsychoses have been placed is in point. I have placed, in the foregoing grouping, general paresis first because it seems to me that the means for its diagnosis are more exact and reliable than the means for the diagnosis of the other forms of syphilopsychosis.

I have placed juvenile paresis second, hoping that the systematic examiner of cases of this group will consider very early in his logical work the question of congenital neurosyphilis. It has seemed to us at the Psychopathic Hospital that a good many errors in diagnosis have been made by the lack of consideration of congenital factors. These errors do not stand out so strongly in district state hospital material as in Psychopathic Hospital material.

The third genus or group of genera under the syphilopsychoses is constituted by the nonparetic forms. Despite the difficulty of the mutual differentiation of this group, I am inclined to separate the genera as indicated into meningitic, vascular and gummatous. To define a genus through negative features is a device which should not be resorted to except in extremity. Accordingly, I hold that the diagnosis cerebral syphilis, cerebrospinal syphilis, as made in many of our clinics, is as a rule no more exact than the more general diagnosis neurosyphilis. When this diagnosis is rendered, there are often no prognostic data available. As a matter of fact, as pointed out by Solomon and myself in the book previously mentioned, much damage may be done to a patient by terming him either general paresis or cerebrospinal syphilis at a time when it is strictly impossible to tell to which genus of the order syphilopsychoses the patient really belongs. At a little later stage in diagnosis, when more data have been collected, it is

virtually always possible, especially with the laboratory data now available, to indicate whether one regards a case as meningitic, vascular or gummatous. Why then, should we stop with the diagnosis "cerebrospinal syphilis," which amounts to little more than the statement that a man has either syphilopsychosis or syphiloneurosis, when we can profitably permit ourselves a generic diagnosis which may indeed practically help the patient a good deal.

Accordingly, I hold that general paresis, juvenile paresis, meningitic, vascular and gummatous syphilopsychoses form fairly well recognized genera in the order of syphilopsychoses. I do not propose a nomenclature, however, for these genera, hoping to excite a critique on the matter.

In addition to these five more or less readily distinguished genera under the order syphilopsychoses, there are a number of less common ones.

Shall we term syphilitic feeblemindedness a form of feeblemindedness or shall we term it a form of syphilopsychosis? According to the general principles of diagnosis by exclusion in order and in the pragmatic and therapeutic interest of the patient, I very much prefer to have the disease classified under the syphilopsychoses. Order Number ii, that of the hypophrenias, is made to include practically all kinds of feeblemindedness which have been defined. Why then should we not speak of a hypophrenia syphilitica? Would it not help the specialists in feeblemindedness so to classify their material? From that more limited standpoint I should agree that hypophrenia syphilitica might be a proper term for the somewhat rare disease, but from the standpoint of neurologic clinics, neurologic and psychiatric clinics, district state hospitals, psychopathic hospitals, I would still think it best to insist on the pragmatic side of the situation by regarding this disease as one amongst the syphilopsychoses. It might be termed neurosyphilis hypophrenica.

Identical considerations hold for syphilitic epilepsy; in fact, it seems to me that the considerations are here stronger; for it is certainly much more definite to term a condition neurosyphilis epileptica than it is to call it epilepsia syphilitica. From the more limited standpoint of the epileptologist, of course epilepsia syphilitica may approve itself, but epilepsy is so much broader and vaguer a concept that it seems to me highly worth while to place all cases of epilepsy regarded as syphilitic in origin amongst the cases of neurosyphilis.

I called attention in the foregoing to one of the war cases in which the acquisition of a syphilitic infection brought out an epilepsy: that case presumably belonged neither in the syphilopsychoses nor in the epileptoses, but rather amongst the psychogenic cases which we rele-

gate to a much lower place on the scale. Such a case might very possibly be classed in the genus hysteria, of the order psychoneuroses. If we hold the diagnostician down in such a case to an exact definition of what he means by making him specify the genus or order in question, we shall greatly improve our logical technic in diagnosis. For instance, is the case one of syphilopsychosis epileptica? Then we would suppose that the spirochetes were in some way acting on the brain so that a true epilepsy hardly distinguishable from sundry other organic forms was being produced. Or, is the case one of hysteria epileptica or hysteria epileptoides in which the adjective conforms with the degree of doubt concerning the observed phenomena themselves? Under the latter circumstance a quite different genesis is to be suspected at work. But, you will reply, how often are we unable to tell which form of genesis is in play? Quite right, one must reply, but until one knows what form of genesis is in play, the true or indicative diagnosis, the really pragmatic diagnosis which will help treatment, has not been rendered.

It seems to me that the diagnostic sheets and statistical tables of many clinics are full of these hedging diagnoses.

As for other less common genera, tabetic psychosis and syphilitic paranoia, something has been said in the foregoing concerning tabetic psychosis (note again that we do *not* mean by tabetic psychosis that subform of general paresis called tabo-paresis); and I shall not delay on syphilitic paranoia, an exceedingly rare genus if it at all occurs.

Under the term atypical, as under other orders of mental disease, I propose to leave room for syphilitic mental diseases of doubtful or hitherto undefined nature, for it is no part of the present endeavor to enumerate and fixate a nomenclature for the psychoses. As in several places stated, I am simply trying to take the groups which modern clinics recognize and place them in a practical diagnostic sequence.

II. Hypophrenoses (the feeblemindednesses, including graded forms of idiocy, imbecility, moronity (in the English nomenclature feeblemindedness proper) and subnormals):

[Syphilitic]

Encephalopathic:
Microcephaly, hydrocephalus, focal brain.

Glandular:
Cretinism, infantilism, dysadenoidism, mongolism (?).

Hereditary:
Feeblemindedness, amaurotic family idiocy.

Atypical.

I have placed the syphilitic group, which might possibly be regarded a good genus, under the hypophrenias in brackets. These brackets here and elsewhere are intended to indicate that the genus has been suffi-

ciently covered in the higher group to which the orderly diagnostician will have already had access.

Refer to what has preceded for notes on whether we should prefer neurosyphilis hydrophrenica to hypophrenia syphilitica. The decision is a close one. I regard it as in the practical interest of the patient to have him classified under the syphilopsychoses. One example of this sort in which an ordinary form of feeblemindedness was found due to syphilis has been given in the Southard-Solomon collection previously mentioned; also in the Waverley Series on the Pathology of the Feebleminded there are data which indicate that we must take into account more than in the past the question of the relation of syphilis to feeblemindedness.

As for the nomenclature of hypophrenia, I have drawn up the arguments for the use of the term hypophrenia as against several others in the literature in a special paper which I hope will be shortly published, entitled, "Hypophrenia and Hypophrenics: Suggestions in the Nomenclature of the Feeblemindednesses." (*Mental Hygiene*, in press.)

Passing to the genera themselves, I am inclined to think that the encephalopathic, the glandular and hereditary groups ought to be regarded as suborders or collections of genera rather than as genera themselves. I do not here propose to suggest a nomenclature for the genera themselves, but have picked out microcephaly, hydrocephalus, other forms of focal brain disorder, cretinism, infantilism, dysadenoidism, mongolism, amaurotic family idiocy and the common form of hereditary feeblemindedness as suitable genera in the present phase of development of the theory of the feeblemindednesses.

With some doubt I place mongolism under the glandular diseases because many workers whom I have met feel that this disease will prove to belong there.

As for the common hereditary form of feeblemindedness, which might be named hypophrenia hereditaria, I feel that it will bulk much smaller than specialists have recently given us reason for supposing. If the encephalopathic cases are pulled to one side (regardless of their possessing tainted heredity, since it is obvious that other factors than mere hereditary germ plasm factors must have been at work), and if many of the glandular cases are set to one side as being directly due to sundry nonhereditary factors, the number of cases which we should be entitled to call hypophrenia hereditaria will be greatly diminished. A number of theoretically preventable cases of feeblemindedness and a number of cases due to brain-destroying and body-destroying factors of a nongerm-plasm nature have been defined in recent work. Of course, the anatomists and pathologists will give

statistics that are possibly unfair to the hypothesis of germ-plasm heredity, since the anatomists and pathologists may overvalue sundry of their brain and body findings; but with all due allowance for this anatomic prejudice, certainly the number of cases of hereditary feeblemindedness in the sense in which we use the term hereditary in the rest of medicine, is year by year diminishing with the progress of medical science.

In my paper of last year entitled, "A Key to the Practical Grouping of Mental Diseases," I endeavored to divide the hypophrenias into genera according to the quantitative results of mental tests. I am inclined to think, however, that this suggestion, however compatible with the spirit of the times with respect to the increasing accuracy of mental tests, is unsuited to the practical work of a clinic. After all, the question whether a patient is a mongolian hypophrenic is more important than whether he is an imbecile or an idiot. The same holds true for hydrocephalus and in fact for a majority of the hypophrenics. The procedure would be to determine your genus and estimate the amount of intelligence shown by the particular example in hand.

As under Group I, I have made provision by the term atypical for genera of an unknown or undescribed nature.

III. Epileptoses (the epileptic group):

[Syphilitic, Group I]

[Feeblemindedness with epilepsy, Group II]

Alcoholic	Idiopathic
Traumatic	Equivalent
Encephalopathic	Narcoleptic
Jacksonian	Borderland
Symptomatic	

Concerning the bracketing of the syphilitic and feebleminded forms, refer to the remarks under Group II.

I will not here attempt to justify the selection of genera under the epileptoses. This is a veritable mare's nest in classification and the man who wishes to use a classification by putting the elements in order of consideration is greatly at a loss. Practically it has seemed to me that if one could push on one side early the alcohol and traumatic question that one would come down on the questions of brain tumor, etc., with a great deal more confidence than if one started in with the latter. Also, practically there are many questions concerning the proper classification of all sorts of diseases having convulsions. The pragmatic answer to the question whether a given disease should be classified under epileptoses or under some other group depends, it seems to me, on the kind of treatment which you propose on your basis of analysis to give the patient. If the kind of treatment is nothing but the regimen, custodial or otherwise, which you prefer for epileptics in general, then the case should be classified amongst the

epileptics. If, however, the convulsions are incidental in some bodily disease, or even in some brain disease in which special surgical treatment or other special treatment may be indicated, then it seems to me that we do the patient a pragmatic injury by classifying him among the epileptics and not in some more definite group of diseases. On this line refer to the remarks concerning epilepsy in syphilis under Group I.

The thumb rule would be: Never classify a case as epileptic if you can be more definite as to its nature and especially its cause.

IV. Pharmacopsychoses (the group of mental diseases due to alcohol, drugs and poisons):
- [Epileptic, Group III]
- Alcoholic
  - A. Pseudonormal:
    - Drunkenness, pathologic intoxication, dipsomania
  - B. Peripheral-Central:
    - Delirium tremens, hallucinosis, Korsakow, pseudoparesis.
  - C. Central:
    - Jealousy-psychosis, paranoia (?), dementia
- Drug:
  - Morphin, cocain, alkaloid
- Poison:
  - Lead, gas, mercurichlorid, special

I will not pause to discuss the details under Group IV. It would seem to me that the designation pharmacopsychoses is a good one, as the Greek word on which the term is founded can be used for both drugs and poisons.

A great deal of theoretical interest attaches to the nature as well as to the diagnosis of the subforms of alcoholic psychoses. I have cast these into three groups, rather inadequately termed pseudonormal, peripheral-central and central. My point is that ordinary drunkenness and so-called pathologic intoxication and dipsomania form three conditions which are, if not normal, then distinctly less abnormal than the other diseases. Drunkenness, it may be stated, is not a form of insanity, and many legislators have so determined, but that drunkenness is not a kind of psychosis I think hardly any one would deny. Here is an instance in which the distinction between a mental disease and insanity comes out very clearly.

But is it possible to distinguish the peripheral-central group from a central one? Practical workers, it seems to me, would agree that delirium tremens, alcoholic hallucinosis, Korsakow's disease and the so-called alcoholic pseudoparesis (if this latter disease at all exists) more closely resemble one another than they do in any of the other forms of alcoholic mental disease. If some one could provide a good designation for this small fraternity of alcoholic disease genera which I have called peripheral-central, he would help our practical work a

good deal. I find a good deal of almost useless discussion in early phases of observation of alcoholic cases as to whether they are instances of delirium tremens or alcoholic hallucinosis. I do not wish to deny a generic value to the distinction, but if we could halt our diagnostic process at the point where the observations stop, we should help psychiatric diagnosis not a little.

The third group that I have termed "central" is composed of the jealousy psychoses which most workers acknowledge that they find in certain instances, paranoia (a much more doubtful matter) and dementia. Here are diseases in which the peripheral element, histologically and symptomatically, is far less in evidence. To be sure there may have been some element of a peripheral nature in the disease at some time, but the chances are that such cases with strong peripheral element belong in the peripheral-central group rather than in the central group. An exact and elegant nomenclature would be a bonanza for practical workers among the pharmacopsychoses.

V. Encephalopsychoses (focal brain lesion group of mental diseases):

[Syphilis]*
[Feeblemindedness]*
[Epilepsy]*
[Alcohol, gas]*

Traumatic. Note that the traumatic neuroses, although they form a group of mental diseases, belong not here in Group V, but below in Group X, the psychoneuroses.

Neoplastic.

Infectious. The infectious group of encephalopsychoses here listed refers to cases like brain abscess and meningitis in which the organism has produced local destructive effects in the brain.

Vascular. Under this group would fall the great group of arteriosclerotic dementias which, be it noted, are parted out from the old age psychoses; Group VIII, below.

Degenerative.

VI. Somatopsychoses† (the so-called symptomatic group of mental diseases):

[Glandular feeblemindedness]
[Symptomatic epilepsy]
Infectious, e. g., typhoid
Exhaustive, e. g., puerperal
Metabolic, e. g., cardiorenal
Glandular, e. g., thyrotoxic
Pellagrous

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*These have been classified, respectively, under syphilopsychoses, Group I; hypophrenoses, Group II; epileptoses, Group III, and pharmacopsychoses, Group IV.

†The term "somatic" is here used following a frequent neurologic plan which employs the term "soma" for the body at large, as against the "encephalon" or brain.

I have tried to define the genera under the five subgroups here mentioned, though I assume that the progress of science will show that a symptomatic psychosis due to the typhoid bacillus is to be distinguished from a symptomatic psychosis due to the pneumococcus; but these are matters for the future to decide.

In practice one should not term a case infectious psychosis, in my opinion, unless an organism has been cultivated from the case or unless there is exceedingly strong evidence that an infection is in play. A good many puerperal cases, when organisms are cultivated therefrom, become on this basis infectious cases rather than exhaustive cases; but who would say that such a translation from one group to another would not be of benefit to the case.

Many authors speak of a toxic-infectious group, of an infectious-exhaustive group or even of a toxic-infectious-exhaustive group, but it seems to me with these double and triple designations we get on not much better than if we confine our statements to saying that the case belongs among the symptomatic psychoses. In short, we are making a very rough diagnosis and placing a case in a large group, but we are rather deluding ourselves that we are making entitative diagnosis.

When infection is not in play and when exhaustion is not in play, I can hardly see the advantage of using the term toxic. The term toxic suggests to the medical hearer that there may well be a toxin in play, that is, such a substance as may be demonstrated in the test tube or under other strictly scientific rules. If pinned down to the meaning of the term, the physician is apt to be reduced to saying that the term toxic refers to certain clinical symptoms that resemble those that are the known effects of toxins or poisons, infectious or otherwise. But is not this a retreat to ground altogether too general to be of value in diagnosis? Perhaps others will not agree with me; but when I see the term toxic and feel that there is no possible laboratory approach to the toxin-poison question, I fall into a marsh of doubt.

The third group of genera here termed metabolic is also sometimes laden with the term toxic, in fact, possibly the term autotoxic might be preferred by many to the term metabolic here used. I can see that the term metabolic is too general a term, but, on the other hand, the term autotoxic seems to specify too much.

The point in the ordering of these subgroups is that, in practical diagnosis, one ought to exclude in succession conditions in which there is a known infectious agent, conditions in which an exhaustive state without known infection, conditions of a general metabolic or autotoxic nature. Those ought to be eliminated from the scene before the glandular cases are brought under consideration.

Possibly the pellagrous group might be placed first under the symptomatic group. Indeed, in regions where pellagra is infrequent,

now and then grave errors of diagnosis have been made. I well remember that one of the first cases of pellagra which came to the Psychopathic Hospital was one of an obscure kind of depression with apparently a cyanosis of the hands regarded as a very proper vasomotor by-effect in his psychosis. By the systematic sequential consideration of these conditions, including pellagra, the question was definitely raised concerning this man whether he might not be pellagrous. The psychosis was then more carefully examined and sundry other features were brought into alignment with the manual lesions. A tentative diagnosis of pellagra was made and the patient thereafter developed a classic form of the disease.

VII. Geriopsychoses* (the presenile-senile group of mental diseases):

[Epilepsy]
[Vascular]
[Alzheimer's]
[Progeria]
[Late catatonia]
[Involution melancholia]
Presenile psychoses
Senile dementia
Presbyophrenia
Senile psychoses

One of the peculiar advantages of this pragmatic sequence of consideration is that the senile dements are removed so far from the arteriosclerotic cases. (Refer to note under Group V.) Kraepelin rightly terms the presenile division of psychiatric cases the darkest field in psychiatry. I am aware how many subgroups Kraepelin has proposed among the preseniles, but for the moment am unable to define what types should be given under the heading presenile psychoses.

VIII. Schizophrenoses (the dementia praecox group):

Hebephrenia	Schizophasia
Catatonia	D. praecoccissima
Paranoid	D. simplex
Cyclothymoid †	Paraphrenia

* This term is adopted provisionally as against the possible term presbyopsychoses, because of Nascher's choice of the term "geriatrics" for his proposed branch of medicine, dealing with the diseases of old age.

† This genus, if it be such, is devised to include the practically very important group of cases in which the schizophrenic process is precipitated by phenomena that resemble manic depressive psychosis, or in which there is a definitely cyclothymic course in itself suggesting the true cyclothymoses.

As for Group VIII, no discussion need be given concerning hebephrenia, catatonia and paranoid. To be sure, concerning the latter Kraepelin has endeavored to distinguish two forms, mitis and gravis, but whether this is a pragmatic distinction of great importance to the future of the patient is doubtful.

As for the term cyclothymoid, I feel that this concept is of some value. First, concerning the term cyclothymoid. The name of this genus, if it be such, would be "schizophrenia cyclothymoides." The ending *oides* used in the specific adjective would be in general borrowed, as in this instance, from some other genus or group. By "schizophrenia cyclothymoides" we would then mean a dementia praecox that somehow very closely resembled a manic-depressive psychosis, that is, a schizophrenia that somehow closely resembled a cyclothymia. If now there were a true cyclothymia (that is, manic depressive) that closely resembled a schizophrenia, we should be forced to dub it "cyclothymia schizophrenoides," borrowing for our specific adjective from another genus and adding the ending *oides*. This procedure would be roughly in accordance with botanical procedure. It would be purely a question of fact whether there is such a condition as "cyclothymia schizophrenoides."

As for the existence of cyclothymoid types of schizophrenia, there can hardly be any doubt that these forms exist. When Kraepelin expanded his original three forms of dementia praecox to nine, he found himself with three new subforms that I have here lumped together under the heading "schizophrenia cyclothymoides." There can be no practical doubt of their existence.

As for the other subheads under the schizophrenias, schizophasia is a small group of Kraepelin's own, of which we now and then see examples. I have added dementia praecocissima group of de Sanctis not because its existence is necessarily well established, but because there seemed to be cases which might well belong in the group if they could be held under observation for some decades longer and their course made out.

It is a question whether dementia simplex should form a genus alongside hebephrenia and whether dementia simplex is more than a mild form of hebephrenia. The term is useful for those cases of slight deterioration which we see in subjects that remain sufficiently well to be self-supporting and only slightly eccentric or dull.

The genus paraphrenia is as Kraepelin has proposed, practically Magnan's disease, that is the *délire chronique à evolution systematisée*. Kraepelin gives four subclasses of this disease which may possibly be species or varieties, namely, paraphrenia systematica, confabulans, phantastica, expansiva.

IX. Cyclothymoses (the manic-depressive and cyclothymic group of mental diseases):

Cyclothymic constitution	Mania
Manic-depressive	Mixed
Melancholia	Involution-melancholia

As to the distinction between manic-depressive and the mixed forms of cyclothymia, I would suppose it wise to call manic-depressive cases (in this generic sense) those in which both mania and depression in different phases of the patient's course are developed.

It would be wise in my opinion to replace the term manic-depressive as a group designation with the term cyclothymia, which brings out the affective features and the phasic features of the disease. If a case is cyclothymic, we shall be able to arrive at the diagnosis having excluded all its competitors for preference down through the schizophrenias.

Now let us say that we are confronted by a case of pure mania or pure depression which we know is not syphilitic, or alcoholic, or symptomatic of some somatic condition, or schizophrenic. We shall be entitled to term it cyclothymic with a high degree of probability, unless perchance on further investigation we determine it to be a psychoneurotic phenomenon. But, again, can we say that this phase of mania or depression is going to be followed by its opposite, depression or mania? It seems to me that we decidedly cannot. The prognosis would better be confined to saying that emotional disorder is likely again to occur. Is not this approximately the extent to which one can now go in making a prognosis in cyclothymic cases? The future may do more for us than has the past. Wernicke remarks that no case of chronic mania was ever initiated by an acute mania. A number of important and easily manageable statistical researches could be made on this line; but psychiatrists are not particularly interested in such statistical researches, however valuable in prognosis their results might be, because they seem to be under the spell of the idea "manic-depressive." According to my conception, the idea of manic-depressive is the idea of a large group of diseases. It is questionable whether Kraepelin discovered a new disease. He defined a great group of diseases, each of which had already been defined, as having certain affinities with one another.

As for the term mixed, I wish by this term to signify cases in which depressive and maniacal phenomena are commingled within a single phase of the disease.

As for involution-melancholia and its placing among the cyclothymias, I do not wish to take a definite stand. Very possibly this disease would better be placed amongst the old age phenomena, as the term involution would suggest.

X. Psychoneuroses:

Hysteria
Neurasthenia
Psychasthenia

This is not the place to discuss the genera and species and varieties of the psychoneuroses. Walton some years since insisted on the value of not making generic diagnoses of neurasthenia, psychasthenia or hysteria. He would have the diagnostician confine himself to terming the case psychoneurosis.

Regarding hysteria, I am inclined to think that in many early phases of these psychoneuroses, Walton's plan is beneficial. It is a question how far a diagnostician wishes to go. Some physicians are perfectly content to call a case mental, that is to say, under the *morbi mentales*, and let it go at that. Others will be content to place a case, for example, under the psychoneuroses and then call in some person especially qualified to cure the case; for the psychoneuroses form essentially the psychotherapeutic group. The specialist may wish to go farther and identify the genus or species, or even the varieties of the large group. No doubt the progress of science depends on further developments in these directions, provided that these developments be pragmatic ones in the interest of helping the patient.

XI. Psychopathoses (the psychopathic group of mental diseases):

Prison psychoses	Sense deprivation psychosis
Folie à Deux	Monomania
Litigation psychosis	Psychopathia sexualis
Paranoia	Psychopathic personality

Concerning the last or eleventh group, there might be much to say. Let me say here that I would speak of this group in common parlance as the psychopathias, not using the ordinal term psychopathoses except in contradistinction to other ordinal groups. The existence of these scientific terms having relatively exact distinction should not preclude our every-day use in the clinic of commoner terms. Just as one would not order *Rosaceae* at the florist's or *Leguminosae* at the grocer's, so one would not use these scientific terms except when one was in doubt exactly where a case ought to belong. In the progress of psychiatric science, the genera under this eleventh group ought to become more and more definite. Some of the genera will doubtless be relegated to pre-existent groups; others may form new orders suitable to elevation to the rank of groups like the psychoneuroses, the syphilopsychoses, etc. I have given in the preceding a small collection of these doubtful psychopathias. None of these require special mention here, perhaps.

Paranoia, I place among the doubtful psychopathias because I do not see that it has been proved to have a schizophrenic nature, and feel that it cannot otherwise be placed in the previous groups. The suggestion that it is a sort of intellectual infantilism is an attractive one, but it seems a little far fetched to place our apparently complex paranoias amongst the feebleminded.

Some persons might object to the use of the term monomania, but if we do not use this term we should need to enumerate such genera as kleptomania, pyromania and poriomania (*Wanderlust*). The polemic in which the term monomania was overthrown is long since reduced to ashes. The term it seems to me remains a good one for precisely those nonsexual cases with unusual development of particular instincts.

As for the term psychopathic personality, it is surely a bone of contention; but if we exclude the sexual cases under the term psychopathia sexualis and exclude the cases with special instincts in strong relief (the monomanias), we shall then have on our hands certain cases of psychopathic personality that are apparently worthy of a place. Many of the so-called defective delinquents very probably fall in this group, though an endeavor should constantly be made to place them amongst the hypophrenics, the epileptics, the schizophrenics, the psychopathic monomanias, etc. All psychiatrists agree that we should not prejudge the situation in criminology by terming all defective delinquents forthwith psychopathic personalities. Let us leave room for the existence of criminals that are not psychopathic.

One might inquire whether there are not certain psychogenic cases that might belong in this eleventh group, that is, cases which cannot be regarded as hysteric, neurasthenic or psychasthenic. Doubtless the neuropsychiatry of the war will help to resolve that question.

SUMMARY

In this paper I have tried to amplify the key to the practical grouping of mental diseases presented to the American Neurological Association in 1917. I have amplified it by proposing certain genera comprised under each of the eleven major groups of mental diseases. These genera have been placed in the sequence supposed to be the pragmatic sequence in which the inexpert diagnostician should seek to exclude successively the various genera; in short, just as the key to the practical grouping of mental diseases dealt in a certain sequence with eleven major groups, so here the diagnostician is given an idea as to the proper method of considering one after another the genera comprised in each great group. No endeavor has been made to revamp or especially modify the ideas of psychiatrists as to what psychotic entities exist. Finality cannot be hoped for either theoreti-

cally or practically. The principle of diagnosis *per exclusionem in ordine* is the special principle insisted on. *It is applicable to any diagnostic problem after the data of observation are collected.* True diagnosis can only take place after sufficient data are collected, and efforts to make diagnoses early in the stage of collecting data are apt to result in prejudice.

The writer earnestly hopes for critique of his propositions. Such critique he hopes will be separated into:

(*a*) Critique of the general principle of *diagnosis per exclusionem in ordine*.

(*b*) Critique of the genera chosen for the different groups.

(*c*) Critique of nomenclature.

But judging from the world's experience in the past, it is unlikely that many persons will be able to distinguish nomenclature from the objects named and the method of using a classification from the classification itself. Herein some nomenclatural suggestions are made; but they have nothing to do with the main line of argument. Herein a certain classification is adopted, but there is absolutely no pretence to originality therein. The writer's main emphasis is on the pragmatic principle of diagnosis, namely, the principle of diagnosis by exclusion in order which principle will prove useful or useless without regard to the classification which it endeavors to exploit or the nomenclature which it uses by the way.

LVII

DIAGNOSIS PER EXCLUSIONEM IN ORDINE: GENERAL AND PSYCHIATRIC REMARKS*

By E. E. Southard, M.D., Boston, Mass.

IS there any excuse for a communication on the general theory of clinical diagnosis? Should not one in the present concrete state of medicine apologize for any generalized or theoretical discussion of a topic which has long since passed into the realm of the dead? If medical schools rarely nowadays, and only as a *curiosum,* deal with the sometime familiar disciplines of so-called medical Propedeutics and the Logic of Medicine, is it not for the very good reason that medicine no longer requires any propedeutics or any special overhauling of its logic?

I have two possibly tenuous excuses for the attempted resurrection, namely, first, that it is a canon of mental hygiene to get in imagination as far away as possible from our terrible environment; and secondly, that the realm of dead notions somewhat well befits a pathologist who has gone into psychiatry.

Speaking to a group of eminent clinicians, I need not insist that I myself make no claims to being a diagnostician of the first water. But just as more or less valuable books on elocution are written by persons without color or pretence of oratory, so perhaps one who is not an especially good diagnostician may descant humbly on the matter. Some five years ago, when I became director of the newly founded Psychopathic Hospital in Boston, it became for the first time my duty to deal concretely with a large series of clinical diagnoses. To be sure, before that time, and in fact, in the year 1909, I had been charged with the task of supervising the clinical and pathologic research work of the state institutions for the insane of Massachusetts, and had already at that time begun to agitate upon the manifest errors in diagnosis which appeared in a small but definite minority of cases of mental disease in these institutions, as tested by the stern criteria of the autopsy table. I took an especial interest in errors of diagnosis in that mental disease entity, about which we know the most, namely, general paresis,[1] and found that one of the most active and thoroughly educated state hospital staffs in the country, namely, that of the Danvers State Hospital,[2] was none the less able to make an error of anywhere from 5 to 15 per cent, according to the fineness of the criteria adopted in the disease general paresis. After all, this work and sundry other articles by my associates and myself[3,4,5,6] remained quite upon the theoretical or unapplied level.

Suddenly, in 1912, I was translated to a region in which exceedingly rapid, not to say provisional, or not to say "snap" diagnoses had to be rendered in what almost any one would concede was the most difficult field of clinical diagnosis, namely, the diagnosis of mental disease. When it is remembered that the task at the Psychopathic Hospital is largely the temporary care of acute, incipient, and curable cases together with a consideration of all the most du-

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bious cases in the community,[7] it will be seen that the problem of a reasonably accurate diagnosis in these cases is not at all easy. It is necessary to remember, also, that our task is not that of the alienist so much as that of the psychiatrist, employing these terms in the differential sense recently advocated in a paper on the nomenclature of mental hygiene.[8] It is not the task of the Psychopathic Hospital to determine, except in a minority of cases, the committability or certifiability of its patients. Of the first 5000 discharges from the Psychopathic Hospital, over 1700 were discharged as "not insane," a term which signifies, not that the patients had recovered, but that they were regarded as not having been insane during their stay at the hospital (the recoveries are over and above the 1700 "not insane"). In short, the task here is one of securing a basis for treatment and counsel for cases that lie often entirely without the purview of governmental control as mechanized by the probate courts. A large part of our problem is medical in the strict sense and not medicolegal.

I may be pardoned for insisting upon this peculiar and almost unique feature of the Psychopathic Hospital work in Boston, because not even my colleagues in the specialty of mental diseases have readily grasped the point. *Alienists* are a type of physician specializing in medicolegal practice, and their task is to determine alienation; that is, the theoretical or practical committability of their cases in the interest of the public welfare. *Psychiatrists* are a type of physician specializing in mental diseases on a much broader basis, namely, the basis of finer diagnoses, involving groups of mental disease and defect, and the psychopathic trends which fall short of defined psychoses and even the quicksand topics of eccentricity, crankiness, and oddity. *Alienistics,*—a medicolegal field,—is pretty sharply distinguished from practical psychiatry,—a medical field. A good psychiatrist ought to be able to accommodate himself to governmental groups well enough to be a good alienist; but, on the other hand, many a good alienist who has dealt for years with the category of committability only, is unable quite to see the point of a diagnosis of "Psychotic, not insane," or of "Psychopathic, not insane." The question of nomenclature is subordinate; but aside from the question of nomenclature, if the practitioner can not grasp the distinction between insanity in the medicolegal sense and mental disease in the medical sense, he is a man unable to keep pace with the modern progress in psychiatry.

What, we may ask, has the question of psychopathic hospital diagnosis of psychoses that fall short of insanity to do with the topic assigned for this communication? How does this work fit into a discussion of the general theory of clinical diagnosis? Psychiatrists have always taken a good deal of interest in classification. I will remind you only of the eminent instance of Pinel, who in 1809, published a *nosographia methodica,* which is one of the best-known of the old nosologies, a very good general view of which is given in Hosack's publication in 1819.[9] I am afraid, however, that most modern practitioners, and especially in America, would be apt to deride it as out of date, if not fundamentally beside the point of these adventures in classification. Of course these scoffers have the entire history of science against them in their scoffing. But the modern practical fellow, especially the pragmatic American, little recks such a

small matter as consistency with the general history of science. Nor will I attempt to defend the various excesses of nomenclature which my psychiatric superiors, both in point of time and in point of capacity, have sometimes perpetrated. It is another queer way which Americans (and I suspect other moderns) have, namely, to confuse the topic of nomenclature with the topic of classification. Classifying for these persons means assigning a name. To be sure, a slight re-reading of that apostle of clarity, John Stuart Mill, would perhaps set these confused minds aright. Still I suppose it is too much to ask of them to re-read even a few chapters of Mill's *System of Logic*.

What, after all, constitutes diagnosis? I do not know whether the Harvard Medical School curriculum is less logical than those of other medical schools that the American Medical Association terms *Class A*, but it is very hard to get from a Harvard Medical School student at any stage, even immediately after securing his M.D., an adequate definition of the terms *diagnosis, symptom,* and the like. Yet would any one of us suppose that these terms can be safely dispensed with in practice or in theory? And even if you could somehow show that a pragmatic view of medicine might throw overboard altogether the distinction between disease entity and disease symptom, nevertheless the books are full of these terms used in a variety of ways, and I am sure it ought to be the duty of every medical course to offer some pabulum (whether propedeutic or by way of appendix) on these matters, which were doubtless comprised in the forgotten and discounted field of so-called Medical Logic.

Many a favorite textbook in the practice of medicine fails to make any point whatever of this distinction, and plumps one forthwith into a discussion,—e. g., of typhoid fever,—jauntily assuming that the student somehow knows by intuition the meanings of the terms, *definition, entity, diagnosis, symptom, symptom-complex, syndrome,* and the like. Perhaps, however, it is better to leave out general remarks in this direction than to put in some which are found in certain other textbooks that have run into many editions. Strümpell[10] makes no special point of the matter but insists in a preliminary note upon the importance of diagnosis, of clinical observation, and of keeping the common courses of all diseases in mind. Here, perhaps, is the most commonplace remark nowadays about diagnosis: diagnosis, namely, is constituted by observation, according to these authors. In fact, there is one textbook (Wilson[11]) that summarily states upon its title page, that the whole art of medicine is in observation. On a later page, it is insisted not quite so broadly, that "It is certainly true that the art of diagnosis is an art of observation;" and again, "The facts of pathology and semiology and the natural history of the diseases constitute the basis of diagnosis." This latter statement seems almost equivalent to the statement that the facts of disease are the basis of diagnosis, which may be in one sense true, but is so little helpful that it might well be left unsaid. Another recent author (Hall[12]) states that "The art of diagnosis consists, then, in gathering all accessible data and arriving at that conclusion which seems the most reasonable as the probable cause of the trouble." This definition evidently goes beyond the former, namely, "Diagnosis is constituted by observation," by considering that the reason is necessary over and above observation. Put in these terms, of course no one of us would care to say that diagnosis consists in gathering

data. Put baldly, it is certainly untrue to say that the collecting of facts constitutes the art of distinguishing amongst these facts. Yet the whole trend of recent work runs in the direction of insisting upon observation at the expense of reason. And, whether or no we teachers are entirely clear in our own minds as to the distinction between diagnosis and the mere recognition of fact as such, certainly our students are not always clear. A student will give the "diagnosis" *fever* or *rose spot,* when he would scoff at making the "diagnosis," *It is raining,* or *The engine is skipping.* If this be diagnosis, make the most of it! Of course, diagnosis is clearly a form of recognition, a form of knowledge, but there seems to be no warrant in raising every elementary form of awareness, kenning, or recognition to the level of diagnosis, which term bears within itself a manifest prefix, suggesting distinction, differentiation, sundering. All my own medical teachers, and I do not doubt the majority of those of my listeners, insisted upon the value of accurate observation. Few of them insisted upon the importance of correct reasoning. Most of them, I take it, partly as a matter of self-defense as they were growing older, were quite sure that maturity of judgment was a thing not readily attained. It never seemed to occur to these teachers that, as the powers of observation were on the wane, the powers of right judgment were to increase. In brief, these teachers, without ever once using the term *logic,* and hardly ever the term *reason,* managed to get into their homilies for students the idea that there was something besides observation, namely, what they called common sense or knowledge of fundamentals, or keeping essential cases in mind, or ripeness of clinical experience, and the like.

Though I believe some course in propedeutics might well be constructed to contain the main principles of the logic of medicine, yet that is not the point of the present argument. Basing my notion upon the practical experience of the last few years, in which I have had contact with clinical diagnosis, always with the autopsy criterion more or less consciously in mind, I want to argue for a more extensive application of the principle of logic in practical diagnosis, and especially the practical diagnosis of the tyro. Perhaps you will think my argument is nothing more or less than a rehabilitation of the diagnosis by exclusion of the older authors. To some extent, the argument does follow that line, though the arguments have been derived from recent practical experience in off-hand and professional diagnosis, and from certain logical considerations. That older argument is found very well expressed in DaCosta's book so long ago as 1864.[13] DaCosta's book, according to an enthusiastic Philadelphian (Wilson[11]) marked an epoch in the progress of internal medicine; and surely many of DaCosta's distinctions are found in several textbooks built upon the lines of his book for many decades after 1864. DaCosta distinguished direct and indirect diagnosis. Indirect diagnosis by exclusion, according to DaCosta "is not on ordinary occasions much employed *nor indeed is it to be recommended*" (italics mine). It is this dictum of DaCosta which I would think ought to be combated in any summing up of present-day processes in diagnosis.

The reasons why DaCosta felt that diagnosis by exclusion was not to be recommended were assigned by him as follows: First, "to prove what a thing is by proving all that it is not, is a very tedious process." Secondly, DaCosta went on to say, "it is difficult to think of all the possibilities." And thirdly, he

said, "pathology is in an imperfect state." Now, as for the latter two reasons, it is clear that the process of direct diagnosis is equally subject to the pitfalls of imperfect pathology, and equally subject to the charge that it is hard to think of all the possibilities. I myself can only believe that these two latter so-called drawbacks are not drawbacks worse for diagnosis by exclusion than for any other form of diagnosis. Accordingly, I believe that DaCosta's objection to diagnosis by exclusion reduces to tediousness; but should we hesitate to say to our students that it is better to be tedious than wrong? The technic of DaCosta's process may be elicited in his own words:

"To cite a case in illustration. A person consults us for a cough brought on by exposure. He has been sick for four or five days, having been previously in good health. We notice, on examining him, that his breathing is hurried, and that he has fever; the lower portion of one side of the chest is dull on percussion, and the respiration there is wanting; the action and sounds of the heart are normal. The facts point to the lung or its covering as the seat of the disorder. We know, furthermore, from the history and the febrile symptoms, that we have to deal with an acute affection. What are the acute pulmonary affections? Acute bronchitis; acute phthisis; acute pleurisy; acute pneumonia. In all there is fever, cough, and impaired breathing. Is it acute pneumonia? No; for, notwithstanding there is in this complaint, in addition to the general symptoms mentioned, dullness on percussion, such as we have here, the dullness is associated with a blowing respiration; whereas in the case before us no respiration is heard. Let us look at the sputum, and see if it is tenacious and rusty colored. It is not; it is thin and frothy. Moreover, the breathing, although hurried, is, when counted, found to be less hurried than it is in inflammation of the lungs. But acute pleurisy may explain all the signs. The patient, too, when questioned, states that he had at the onset a sharp pain in his side; and this we are aware takes place in pleurisy. The vocal vibrations, likewise, are noticed to be absent on one side of the chest, which, when carefully measured, is evidently enlarged. This corresponds in all points with what happens in pleurisy in the stage of effusion. The disease *is*, therefore, acute pleurisy in the stage of effusion. We finish the diagnosis by ascertaining the existence or nonexistence of other maladies, and by taking note of the severity of the complaint; that it has occurred in a young and robust person of good habits; and that the symptomatic fever is very active."

But what is a direct diagnosis,—one not made by exclusion? According to Wilson, "A direct diagnosis is made when the history of the case and the clinical phenomena are sufficient to warrant a positive conclusion." Wilson goes on to say that a diagnosis by exclusion differs from differential diagnosis only in scope. Strange as this phrase may sound, it strikes me that there is a good deal of truth concealed in it. I feel that, so far from admitting with DaCosta, that diagnosis by exclusion is not much employed or to be recommended, practically every diagnosis is by exclusion, whether consciously or unconsciously.

Musser remarks concerning diagnosis that "the data collected are sufficient to warrant a positive conclusion. The direct method is scientific, rational, and most practical. It is a practice of purely inductive reasoning." I am not sure whether it would not be sounder to say that it is a process of purely *deductive* reasoning, but modern logicians have conclusively enough proved that practically all reasoning is both inductive and deductive, so that it would not pay to quibble over the claim that direct diagnosis is "a process of purely inductive reasoning." As to the method of diagnosis by exclusion, Musser remarks, "Thus, a symptom group may suggest several diseases; each affection must be passed in review and excluded until one is found which closely corresponds to the data of the case under consideration." In short, it would seem that diagnosis by

exclusion is an effect of the *force majeure* of complex and obscure data. The battle is easily won when it is a question of perceiving a symptom and suspecting a disease, whereupon all "the data collected are sufficient to warrant a positive conclusion."

Now, possibly it is the complex and obscure nature of mental disease which has caused me to feel that diagnosis by exclusion is a method that requires rehabilitation in the minds of teachers and in the pages of textbooks. In medicine and biology at large in the middle of the nineteenth century, it was the fashion to denounce speculation and extol observation. So many of the medical and biological sciences depended upon the microscope and the visual process, that nothing was more natural than to see great virtue in observation as compared with the vagaries of the *Naturphilosophie*.[15] We are now so far beyond the range of that old *Naturphilosophie* (unless the idea of Pan-Germanism may be regarded as a remote result thereof), that it should be quite safe to uphold the reason as entitled to a seat beside observation in the process of diagnosis. Applied logic accords to observation its full worth. Secondly, a proper value is assigned to what may be inaccurately summed up in the phrase *comparative method*. It is the comparative method probably that is being used in diagnosis for the most part at the present day. That is, one picks a symptom, suspects a disease, constructs a supposititious diagnosis, and proceeds to compare it with all the data of memory or reference books. It is all one, whether you pick one symptom—e. g., the so-called presenting symptom[15] popularized by Richard Cabot,—or assemble a symptom group; the process remains the same, that of matching the data of the case with some supposititious diagnosis. Now in the field of mental disease probably every one of you has had the experience of matching virtually all the symptoms of a case with the majority of those given in the textbooks for several different mental diseases. In short, the comparative or matching process has been only too successful, and the diagnosis might well be rendered by lot from amongst a not very small group of textbook headings.

Of course, the comparative method involves the use of observation also. The distinction between observational recognition of a condition and the comparative determination of a disease by a direct method, is not a mutually exclusive distinction. So also in respect to the third method of applied logic, namely, the statistical method. The statistical method requires both observation and comparison, but entails also certain new features. Probably Richard Cabot has popularized the statistical method of diagnosis more than any other recent author. Cabot finds a presenting symptom which becomes to him a lead. This presenting symptom is often something complained of by the patient, but also is often something noted by the physician that the patient did not subjectively recognize. Cabot has founded a differential diagnosis upon a selection of the most common presenting symptoms. He goes on to list the causes of these symptoms by their frequency, and then, to use his picturesque phrase, "follows the symptom home." Following the symptom home means, naturally, employing all manner of comparative and observational methods in addition to the merely statistical one. Nor do I wish to give the impression that Cabot overemphasizes the statistical method in his account. He merely emphasizes it more than most previous authors. Nor would his account fail to insist that observation is indis-

pensable, for he carefully states that diagnoses are missed usually because physical signs are not recognized. That is, for Cabot, poor diagnosis is usually a matter of poor observation. But occasionally diagnoses are poor on account of poor reasoning, and Cabot's book is an argument for a more comprehensive grasp of the possibilities of diagnosis. Others may insist upon the recognition of the symptoms and signs by proper methods of observation, and Cabot, like many others, has in other works given an account of these matters along approved lines; but he dreams for the purpose of differential diagnosis of a "cash-register of causes." He wants possibilities sifted. He wants clues, radiations, and leadings followed out along lines that are essentially statistical. One proceeds from the presenting symptom to the possible causes; from the possible causes to the probable causes, and from the probable causes to the actual cause. In the technic of this determination, Cabot, as is well known, much insists upon the statistical configuration of the different conditions in which the various presenting symptoms are found.

Cabot notes that physicians in the matter of differential diagnosis "are very suspicious of any attempt to tabulate their methods of reasoning." He speaks of profiting much by Herbert French's *Index of Diagnosis*[16] (first published in 1912), a book which I find is greatly affected by medical students of my own acquaintance. French claims his index to be an index of main symptoms, though there seems to be no internal evidence in the book just how these main symptoms were selected. French, like Cabot, deals largely though not exclusively with symptoms complained of.

It is readily seen that Cabot's ideas (though they would have been readily comprehended by DaCosta in 1864, since they are founded upon the statistical principle which had already been a few decades in vogue in DaCosta's time) would not have appealed especially to him as important in the process of direct diagnosis. Feeling as DaCosta did concerning diagnosis by exclusion as a *dernier ressort,* he would not have seen the compelling value of Cabot's statistical frequency tables for the distribution of symptoms in different conditions. Moreover, it is doubtful whether in 1864 hospital records were so extensively kept as nowadays, despite the fact that intensive records of special cases may have been kept at a relatively higher standard than now prevails in institutions where the real work is done more by interns than by visiting physicians.

The situation, then, is that in some cases what might be called an observational diagnosis can be rendered. Personally, I should feel that the term *diagnosis* was being prostituted somewhat when the diseased condition is recognized forthwith on the basis of some pathognomonic symptom or combination of symptoms. But let the phrase pass; the result of naming the disease and getting a practical basis for treatment is attained by a process of pure observation. The so-called direct method of diagnosis is hardly on a higher plane logically speaking than that of observation in many cases; but in others, and perhaps in the majority of cases, this process of differential diagnosis by the so-called direct method, is one of comparison; that is, of matching the phenomena of the disease observed with those of some classical or textbook type. The unduly maligned method of indirect diagnosis, or diagnosis by exclusion, likewise employs comparison inasmuch as it excludes disease after disease by discovering that not

enough exists to match the types in question. Having risen, accordingly, from a process of direct recognition (*gnosis* rather than *diagnosis* in any logical sense) to a process of somewhat less direct differential diagnosis by a positive matching or a negative excluding process, we finally arrive at a more modern technic of studying the frequency of symptoms in certain large groups of comparatively well observed cases (the majority of Cabot's tables deal with 250 cases analysed at the Massachusetts General Hospital). We notice that the process of observation is indispensable throughout. Extreme exactitude of observation is not perhaps so necessary in statistics as in individual cases; at least errors of observation may be assumed to balance themselves to some degree.

Is there no further sharpening of our technic of diagnosis? In the practical handling of the diagnostic problem at the Psychopathic Hospital, in the course of two years' observation I became interested in the fact that, where diagnoses were not matters of mere observation and direct differentiation, but where processes of exclusion were necessary, I found myself endeavoring to exclude conditions in a certain *definite order.* It is this ordinal principle which I believe might constitute an advance in practical diagnosis, were it to be insisted upon in still other branches of medicine than psychiatry. I have recently made some endeavor to apply the principle to the situation in clinical neurology but shall not here mention the results of that inquiry in any detail. After the first two years of off-hand and provisional diagnoses at the Psychopathic Hospital, I then considered the matter of exclusion in a definite order for some three years before laying down the order in question (see below). I think it would be presumptuous for any one in any special branch of medicine to draw up a definite order of diagnoses in any other. For example, however convinced I might be that diagnoses in dermatology are actually made consciously or unconsciously by some process of successive exclusion of conditions in a definite order, I should not (as I am far from being a dermatologist) have the slightest ground for suggesting an order for dermatological diagnoses, or a list of conditions to be put in order. Not being an internist in the field of cardiac disease (that is to say, not being in any sense an expert with the electrocardiograph and all the rest), I certainly could not advocate any particular order of diagnosis for heart disease, however strongly I might be convinced that such an order must exist.

At this point, I looked into certain works on logic to learn what might be said concerning these different principles of diagnosis, and found in the late Josiah Royce's contribution entitled "The Principle of Order" in the *Encyclopedia on Philosophical Sciences,* an exposition very much to the point.[17]

I presume that most of us have few memories of logic except the syllogistic logic of such college textbooks as that of Jevons. Some of the older amongst us will still have used Mill's *System of Logic,* or textbooks closely modeled thereupon. The more recent advances in applied logic are a closed book to most college and medical students. But in addition to so-called formal logic used as an irregular discipline in most colleges, there is an applied logic or methodology which deals with the norms of thought as applied to the methods of the special sciences. The formal logic of a book like that of Jevons is only a very subordinate part of logic as at present defined. Royce speaks of the new doctrine as

the Science of Order. This science, he says, is today in a very progressive condition, is in some notable respects new, and offers inexhaustible opportunities for future progress. Already in the Socratic and Platonic handling of these topics, the systematic nature of the world's construction, the objectivity of the world permitting inference concerning its various truths, and the order that rational processes show, had already become clear. Types, forms, and relations are at least as real as the facts of the physical world. Aristotle, Bacon, and still more importantly, Galileo, made important advances in logic. Today no one will question that every science in dealing with its facts must employ methods of classification, in no wise different from the method used by Socrates in his dialectics. To be sure, a science which uses only methods of classification must now be regarded as a very young science. Botany and zoology have long since passed the phase in which classification is the dominant interest. Anthropology, on the other hand, is largely classifying still, though there is some tendency to the development of a higher method of logic, the comparative study of the forms and results of human culture.

Royce mentions that, amongst the medical sciences, psychiatry is just now emerging from a stage in which the bulk of the science was made up of the classification of cases, symptoms, and disorders. Psychiatry is now about to work on a higher plane of methods. The more complex the facts, the harder it is for a science to get beyond this first stage of classification.

What are these higher processes of applied logic? There are mainly two: The method of comparison, and the statistical method. The comparative method compares the corresponding stages in the various processes of products of natural evolution in the science in question; whereas the statistical method uses exact enumerations as the basis of its science. Of course, the two methods are not always sharply to be distinguished; whenever one has to compare numerous evolutionary processes, all the results of these processes have to be enumerated. In these instances, however, the statistical method is merely an auxiliary to the comparative method. When, however, we come to such matters as the mortality tables of insurance, the sociology of marriage and divorce, of suicide and crime, or of commerce and industry, we are dealing with topics in which the statistical method is virtually the only one at present available. We study, for instance, how human mortality varies with age; how the science of an organ or an organism follows conditions determined by heredity or environment. As a result of such statistical processes, the statistician becomes able to deal with aggregations or blocks of facts; orders or aggregations which may be treated as units of a higher order.

When sciences become developed still further, these two methods of comparison and statistics are used in a still more intimate manner, combined in a method which Royce terms the *organized combination of theory and experience.* By means of the statistical and comparative methods, we discover laws of nature that have a certain degree of probability, the degrees of which we may trust on the basis of fair samples of the phenomena of the given science. We then construct laws on the basis of hypotheses derived directly from fair samples of fact. Hypotheses thus drawn may be of the simple type directly

refutable or directly verifiable by immediate recourse to the facts; or on the other hand, hypotheses may require much more work for their testing. In fact, the more direct the proof, the more valuable the hypothesis, since the success of the hypothesis would here depend upon the falling-in-line of vast numbers of facts which on no other basis could be supposed to fall in line. To combine hypothesis, theory, and observation, we first draw up a hypothesis regarding the constitution or laws of some region of physical fact. Upon the basis of this hypothesis, we make an extensive and exact deductive theory as to what *ought* to be present if the hypothesis is true. "The more extensive, exact, and systematic the theory thus made possible proves to be, the larger are the possible samples of the consequences of the hypothesis which are available." We then collect samples of facts by means of observation and experiment, and proceed to compare them with results of our elaborate deductive theory. The more elaborate and extensive our theory, the larger the range of facts that we can draw upon for comparison. We are not now restricted to noting what proportion of members of a sample have certain characters. On the other hand, if the deductive theory is a highly developed and elaborate one, we shall find that almost any sample available for comparison is minutely verifiable in detail with some part of the original hypothesis. In short, the value of this method depends upon "the exactness, the order, and the systematic character of the concepts in terms of which the hypotheses thus directly tested are defined." It would thus appear from this highly condensed statement of Royce's view, that "the organized union of theory with observation requires for its perfection concepts and systems of concepts which permit of precise and extended deductive reasonings." It is surprising how small the samples need to be when it comes to a judgment as to the applicability of a highly complex hypothesis. A few small samples taken from widely separated parts of the system will prove or disprove the virtues of the hypothesis.

Without asserting that medicine is yet in a stage to profit like physics and chemistry from this organized combination of hypothesis, theory, and observation, it is clear that many parts of medicine are far beyond the application of mere statistics or of mere comparison, and still farther beyond the modest virtues of pure observation.

Without venturing further at this time into methodology and modern applied logic, it is clear that mere observation, mere comparison of stages, mere enumeration, are not the whole story in any science. If the medical sciences are going to advance in their logic, it is more than likely that their logical advance will take place in the direction of a greater insistence upon order. I hold accordingly that, if Royce's exposition of certain modern developments in logic is a sound one, the next stage of development in medical thinking will be a reduction of its interests more and more to order and system. There is reason to hope that by the reduction of medical as well as any other scientific facts to order systems, a greater and greater success in deduction will be attained. I conclude, therefore, that when I found our process of diagnosis at the Psychopathic Hospital employing more and more the concept of order, I was simply discovering a small fact bobbing in the big stream of logical advance.

In the light of present knowledge, how almost naïve and childish appears the old dictum that disease is life under altered conditions! Here were but two elements: life on the one hand, and change on the other. Each contains a host of undiscovered, undigested facts. How slender a contribution appears the claim that diseases must be instances of either the augmentation, the diminution, or the mere modification without increase or decrease of structures or functions! To be sure, bacteriology shortly yielded a dyadic system of germ on the one hand and host on the other, which for the moment and for a decade or so allowed one to hope that the formula "Disease is life under altered conditions" might be simply rendered into, "Disease is the effect upon an organism of a germ." Then came the contentions of Metchnikoff concerning phagocytosis, and the more complex interrelations of the host with its contained germ.

Shortly, in the nineties arrived triadic conceptions of the interrelation of the organism, the foreign substance, and sundry intermediary bodies; and early in this century was unfolded the still more mysterious situation of anaphylaxis. In short, the problems of medicine are becoming more and more rich and complex, more and more verifiable by small samples of fact taken at widely diverse parts of the system.

The rapid complication of the research situation in medicine at large, as typified especially by the facts of immunology, has its reflection also in the slower moving departments of clinical diagnosis. We saw in textbook after textbook insistence on diagnosis as a matter of observation (going back very far, for example, to Baglivi[18]). We then found, as in virtually all the medical textbooks in the latter half of the nineteenth century, a new form of assurance: The diagnostician is not now satisfied with what he calls direct diagnosis.

The aim here is to enroll the case in some definite group of diseases recognized by pathology. The difference between perceiving that "this fruit is an orange" and scientific recognition of a diagnosis is merely a question of degree. It is not so easy to say that "this is a case of typhoid fever," as it is to say that "this fruit is an orange," because of the more concealed and special nature of the points on which we rest our diagnosis of typhoid fever. The method is one of comparison of certain perceived characters or features with certain signs already known to us as pertaining to certain disease groups. Whether the symptoms are simple, like a friction rub, or complex, like the chemical analysis of a secretion; whether the symptoms are present at the time of examination or are merely anamnestic, from the diagnostic point of view, they are speedily divided into pathognomonic symptoms on the one hand, and more general ones on the other. The tubercle bacillus or the Argyll-Robertson pupil might be mentioned as examples of pathognomonic symptoms in this broad sense; whereas fever and vomiting would be examples of symptoms which belong in a great number of disease groups. But whatever the kinds of symptoms we recognize, the process of diagnosis appears to be one of comparison of those symptoms that we perceive with such symptoms as we know to characterize a given group of diseases, and upon the discovery that the symptoms of the disease in hand correspond with the symptoms of the disease type

which we know from the books or from experience. We draw a conclusion, then, on the basis of similarity of symptoms. We may draw this conclusion from a pathognomonic symptom, which beyond any question indicates the existence of a given disease; or if no pathognomonic symptoms are present, we may ground our diagnosis upon the harmony between the general symptoms in our patient and the symptoms that belong to some described disease; and of course we are throughout well aware how unsuccessful may be a diagnosis based largely on the similarity of general symptoms to those of the books. This method of diagnosis, which we may briefly term the "type-matching" method, is doubtless the one extolled by the older workers under the name direct diagnosis. It represents an advance upon purely observational diagnosis,—that is, diagnosis by inspection,—and is a form of diagnosis by comparison.

Diagnosis by exclusion or by differentiation is a method somewhat decried by older workers and now brought into great prominence by such work as that of Cabot and of Herbert French. Though there is nothing logically new in this work, there is a new emphasis in it. It is still, logically speaking, an application of the comparative method greatly bolstered, as Cabot handles it, by the statistical method.

The general nature of differential diagnosis is somewhat as follows: As it is impossible to consider all the phenomena in a given disease, we first select a group of diseases in which a certain sign is found. We look in the case under observation for some more or less outstanding symptom. This common symptom which we would use as the point of comparison amongst diseases, is called by Bieganski the *index of differentiation* or the difference indicator. Cabot has more recently termed one form of it the presenting symptom. This indicator or presenting symptom is chosen because we by experience know that it occurs in a large number of diseases. To be sure, it is much better not to take a too-general indicator. Thus, in the question of typhoid fever, it would not be so advantageous to begin with fever, perhaps, as to begin with diarrhea. The process of differentiation now begins: We compare the case under observation with those diseases which we have found to be somewhat similar thereto. We pass from one disease group to another. We constantly keep upon one side the observed case with all its symptoms, and on the other the total series of diseases which in a certain respect are similar to it, namely, in respect of the common trait, index of differentiation, or presenting symptom. We proceed to look for differences. The observed case may not belong in a group because it does not possess the symptom which is constant in that group; though the indicator is possessed by both diseases, a symptom constantly present in the compared type is absent in the observed case.

Or the observed case may not belong in the compared group because there is a symptom in the observed case which never occurs in the compared group.

Again, the observed case is not the disease with which we are comparing it because we know that in the compared disease a symptom constantly occurs which is the direct opposite of some symptom observed in the case in hand.

Moreover, there may be an incongruence of symptoms of a quantitative or qualitative nature, a difference which does not proceed to the point of complete absence or perfect antagonism of symptoms.

Following is a table which embodies the process-types of clinical diagnosis now in vogue:

TABLE OF PROCESS TYPES OF DIAGNOSIS

I. Inspection.
II. Expectation.
III. Induction.
IV. Comparison
  (a) Similarity.
  (b) Similarity and difference.
  (c) Difference.
V. Ex juvantibus.
VI. Ex nocentibus.

I may say at the outset that it is the endeavor of this paper, granting each of the three subordinate methods under the head of Comparison to be a separate logical method of diagnosis, to add a ninth method. This ninth method I shall choose to call *"Diagnosis per Exclusionem in Ordine." Diagnosis per exclusionem in ordine* is merely a method of employing logical comparison of Type IV (c) of the above table *in an orderly manner.*

But before attempting to speak of this newly described type of diagnosis I must briefly describe the eight classical methods so that I may, if possible, demonstrate that the ninth or proposed new method has novelty. For the purposes of this discussion I have gone rapidly over a great many of the older textbooks of medicine, studying largely the logical remarks by their authors in the prefaces and introductions thereto. I then examined many of the textbooks in Medical Logic which were for some years the vogue in medical schools. From the study of textbooks, largely in the latter half of the nineteenth century, and from the study of works on Medical Logic I have arranged and combined the methods of the above table. I have been especially aided by the modern work of Bieganski, a Polish work available to me in German translation from its second edition. This work may be referred to for a good summary of the older points of view, together with the fruits of some Polish polemic in which Bieganski had for some years been engaged. The older work of Oesterlen,[20] published in Tübingen in 1852 and translated into English[21] in the Sydenham Series in 1855, is almost barren for modern purposes (so much so that I found the leaves uncut both of the original and of the translation in the New York Academy of Medicine!) Of still older literature, the Medical Logic of Sir Gilbert Blane,[22] illustrated by conclusions concerning yellow fever, is of great interest, though his classification of the elementary principles of life as generative, conservative, temperative, assimilative, formative, restorative, motive, sensitive, appetitive and sympathetic, is an example of categories which, however true and from various points of view feasible they may be, are of little diagnostic value. This older work often fails to distinguish the question of classification from the question of what is to be classified. Sir Gilbert Blane's work upon yellow fever, and especially upon scurvy, derived

from his expert contact with marine diseases in the British West Indian campaigns, is of greater value than his excursions into logic. Blumenbach calls Sir Gilbert Blane the most learned and classical physician of the age; but despite this German appreciation, little has been done in Germany of an original nature on medical logic. Oesterlen appears to be in the general nature of a physician's reaction to the logic of John Stuart Mill. Apparently someone was impressed with the possible value of such work as Oesterlen's, and Francis Ogston was given a new chair in Aberdeen as Professor of Medical Logic and Jurisprudence. In a syllabus[23] of his highly interesting course of thirty-two lectures, he refers to his indebtedness to John Stuart Mill and to Oesterlen, traces the history of the topic from Aristotle to Bacon, thence to Blane and Oesterlen, and comments upon the absurd distrust of theory which he found prevailing in Britain. Ogston called the merest routine practitioner a bold speculator and imperfect observer of facts. He remarks that the limits of pure observation in medicine are soon reached and discusses in separate chapters the inductive or analytic method as opposed to the deductive or synthetic method, rightly holding that the latter is a much more important concern of medicine than the former. Under the inductive or analytic method Ogston considers the processes of observation, comparison and analogy, and the numerical method. Under the deductive or synthetic method he considers first the process of analysis for deductive purposes, then the process of ratiocination and of verification. He discusses, among the processes subsidiary to induction, such matters as simple observation, experiment, hypothesis, analogy, the numerical method, terminology and classification. Ogston denounces the current nosologies as obstructive and inelastic and devotes special attention to what he calls the fallacies of nonobservation as opposed to the fallacies of malobservation. Much of this suspicion of Ogston can be traced in Oesterlen and Sir Gilbert Blane, to say nothing of their masters in logic, John Stuart Mill and Sir Francis Bacon.

Sir Gilbert Blane had, for example, in 1819 spoken of the sources of error in medicine as (*a*) the fallacy and danger of hypothetical and theoretical reasoning as especially demonstrated (according to Blane) in Boerhaave, (*b*) the diversity of constitutions, (*c*) the difficulty of "appreciating the salutary efforts of nature and of discriminating them from the operations of art," (*d*) superstition, (*e*) the ambiguity of language, and (*f*) the fallacy of testimonial.

Much can still be gained, no doubt, from a perusal of such works as those of Sir Gilbert Blane, Oesterlen and Ogston, and still more could doubtless be gained by the physician from a close study of John Stuart Mill and his predecessors in pure logic, but these studies were almost wholly given over with the onset of the evolutionary theory in the late fifties, with the crowding developments of pathology and physiology and especially of the bacteriology of the eighties and the immunology of the nineties of the last century. There has, in fact, crept over us the feeling that logical method is an inheritance of us all in like degree and that whatever we do not know of logic at the outset of medical practice we shall never learn. It is precisely the popular single volume textbooks that fail to yield much of practical value to the medical student seek-

ing to perfect himself in the *logic* of diagnosis. The dictum, especially of Da Costa and of his successors in the manufacture of single volume textbooks, namely, that the art of medicine consists in observation, has apparently abolished the necessity of logic. Carried away by the magic of this word "observation," many authors of physiologic works dealing with the most intricate discussions and fine-drawn conclusions cheerfully called their inductions and deductions by the term "observations." The obvious fact is that diagnosis does not consist in observation at all. Diagnosis requires observation, proceeds upon a basis of observation, but is in itself a process of combination, of reasoning, of calculation, or of some higher kind of intellectual process which takes observation for granted, uses and chooses amongst observations, but in no whit makes them.

Amongst the methods tabulated above I have first set down, following many of the books, *diagnosis by inspection.* Here, at the outset, it may be inquired: "Are we not terming a method of diagnosis what is nothing but a method of observation?" This I freely concede. The names of diseases are achieved by a process of inspection. The result is what we term a diagnosis, but the process by which the so-called diagnosis or disease-name is arrived at is not a process of reasoning at all, but (as stated above) a process akin to the statement, *It is raining,* or *The engine is skipping.* In short, no process of diagnosis in the logical sense is here being used. A diagnosis or disease-name is achieved, but reasoning is not employed. It is only, then, by courtesy that we include inspection under the process-types. The diagnosis follows the inspection as night follows day, or as black follows white, or as man follows woman. The process is one of association of a perfectly simple type. This kind of so-called diagnosis can be made by laymen often as well as by physicians. I am afraid that the ideals of many medical teachers, as they are got by the eager students, consist in teaching them thumb rules of inspection rather than the more tedious and complicated methods of diagnosis. I recalled above how Da Costa spoke of diagnosis by exclusion as a tedious method.

The second method tabulated above is one that I have presumed to term the method of *diagnosis by expectation.* We are familiar with the so-called expectant treatment in therapeutics, but, so far as I know, attention has not been called to the method of diagnosis by expectation. This is, of course, particularly employed in the fulminating, acute diseases on one hand and the chronic diseases on the other. In the one instance there may not be facts enough to permit a diagnosis by inspection and there may not be time enough to permit elaborate comparisons; accordingly, diagnosis goes by the board until an autopsy either is or is not made. As for the slow, hesitating course of certain diseases, the Micawber attitude is still easier to assume.

I suspect that the method *by induction* is commonly employed in a manner not far removed from the process of expectation. This method by induction, unlike the method by expectation, is mentioned in the classical works, and also in the modern work of Bieganski. One concludes that the patient looks tuberculous. Here is the suggestion of a diagnosis by inspection. Perhaps one is an office practitioner and time forbids exactitude. One examines the pulmonary

apex and discovers suspicions of dullness. One requests the sputum and proceeds to a positive diagnosis of tuberculosis on the basis of tubercle bacilli found in the sputum. Here is a successful case of diagnosis by induction by a sort of skipping of intermediaries (to use James's phrase) and by the least possible work. If the diagnosis is successful, well and good! If unsuccessful, one must proceed to more elaborate methods. It is always possible, too, that the patient may have been suffering from a combination of two or more diseases and that the tuberculosis e. g. found may be the lesser of two important conditions. One ought not, and I do not, condemn this method by induction just because it is not time-consuming and just because it does not succeed in all cases. In the hands of the expert the method of diagnosis by induction (as the phrase is here used) and the method of diagnosis by inspection are highly commendable methods. In a pragmatic age one ought not to denounce them. From the standpoint of a medical student, however, it would seem wise not to dazzle him with the brilliancies of inspection and induction (in the above narrow sense), but one ought to expound to him the nature of the more complex methods which he must often use in cases that are themselves at all complex.

The methods of *diagnosis ex juvantibus* and *ex nocentibus* tabulated above are in a sense experimental methods of diagnosis on all fours with the method by induction. The diagnosis of syphilis by the administration of antisyphilitic drugs and the procuring of a cure or an alleviation of symptoms is an example of diagnosis *ex juvantibus*. The provocative Wassermann reaction upon the administration of salvarsan is an example of the method of diagnosis *ex nocentibus*. It would be a mistake to consider the administration of drugs for these purposes therapeutic. The methods are methods of diagnosis purely.

Dismissing the methods of inspection, expectation, induction, *ex juvantibus* and *ex nocentibus,* we come to the methods of *diagnosis by comparison,* to which I hope to point out a small addition or modification which I conceive to be of value. Earlier in my communication I have sufficiently discussed these methods and I pointed out the necessity under which we labor of using the method of diagnosis by exclusion in cases where there are no indicator symptoms or presenting symptoms (to use Cabot's phrase for certain indicator symptoms). I wish to illustrate this method of *diagnosis per exclusionem in ordine* from the field of mental diseases, rehearsing the main groups of a practical key[24] which has been built up on the basis of Psychopathic Hospital data.

In passing, I may say that psychiatrists are far more at one in the matter of diagnosis than the general medical man is apt to believe. The unanimity in point of view is far greater than one would suspect from certain nomenclatural differences. The nomenclature of psychiatry is by no means as fixed at the present writing as the nomenclature of dermatology. Yet the unanimity of psychiatrists concerning the major groups of mental disease is, I make bold to say, almost as great, if not quite as great, as the unanimity of dermatologists concerning their main groups. The specialists in psychiatry have for some years past been much interested in proposals of the American Medico-Psychological Association to secure a thorough and acceptable classification of mental diseases to which the State institutions for the custodial insane could conform. The

classification adopted by the Medico-Psychological Association in 1917 is, in general, a highly acceptable one.[25] Probably no member of the committee charged with making the classification is entirely satisfied with it in details. Some diagnosticians go so far as to say that in the interest of the patient no classification in the sense of the statistical tables of institutions is worth while, since every instance of mental disease is virtually *sui generis*. There is, of course, much truth in this contention, and the good therapeutist never forgets this fact, borne in upon the mind as it is by the tremendous individual variety of the psychopathic reactions of the individual as such. I found that most of the leading American textbooks of psychiatry were also practically at one in the major categories, despite perturbing difficulties in terminology.

The task of the Psychopathic Hospital, dealing as it does not merely with custodial insane but with all the penumbra of "near-insane," of incipient, mild and curable cases, and with many psychoneuroses which would not ordinarily be classed among the insanities, is a somewhat broader and deeper task in diagnosis than that of the custodial institutions for the insane. The Psychopathic Hospital stands as a sort of vestibule between the community and the custodial institutions. From its meshes go back into the community numerous cases whose psychopathia is not far advanced enough to go through the interstices. Not only insanity in the narrow sense, but a variety of other conditions of the *non compos mentis* group, namely, feeble-mindedness, epilepsy, and even many forms of alcoholism come within the purview of the Psychopathic Hospital. The task of rapid diagnosis was one which had never faced the custodial institution for the insane, because to the latter flowed only cases in which diagnostic experts had made their decision outside the walls of the institution and with the concurrence of the Courts. It became necessary, therefore, to contrive more rapid methods of handling cases than had formerly been available, inasmuch as keen and mature experts were not available for the great run of cases (the Psychopathic Hospital ran the number of voluntary cases and of so-called temporary care group of cases up by the hundreds as soon as it was founded). Some provision had to be made whereby younger men could make reasonably accurate primary siftings of the cases. The rapid development of mental hygiene stole away the young men at such a rate from our staff that provision had to be made for shifting staffs of relatively inexperienced persons. The situation was not unlike that which faced the army itself at the outset of the first draft.

Mental diseases have no indicator symptoms, barring a few, and indeed a very few, so-called pathognomonic symptoms. Psychiatry was devoid of pointers, such as would be available to the specialist in the exanthems, for example. In the absence of indicator symptoms, some method had to be chosen by which every case would have every major hypothesis concerning its nosological grouping raised at some time during his hospital stay. The legal details and official regulations were such that these hypotheses had to be raised within a very short period. Early in our experience the laws and regulations were such that some kind of diagnosis permitting proper disposition of the case had to be made within four or five days. This proved to be too brief a period in

which to make proper disposition and the Temporary Care Law, under which the majority of Psychopathic Hospital cases are admitted for observation directly from the community (without court process), was modified so that a period of seven or eight days is now available. Official disposition is at present made in a total period of ten days, instead of in a total period of seven days; but in practice the external conditions of transfer to social adjustment take from two to three days, so that the total time available for group diagnosis is now a period of from seven to eight days. During this period of seven to eight days what can be done?

I assume that in private practice it may not be necessary to secure the Wassermann reaction of the serum in each and every mental case. For my part, I never feel certain of the diagnosis unless this precaution is taken. In any event, in Psychopathic Hospital practice, with the varying conditions of staff and history standard, it shortly proved necessary to get the Wassermann data from the serum in each case. Only a few of the more conservative hospital superintendents failed at first to approve this plan. The Danvers Hospital in Massachusetts and the hospitals in Michigan, under the influence of the Psychopathic Ward at Ann Arbor, had early adopted getting the Wassermann reaction of the serum as routine. Upon reflection, I found that the proper disposition of almost each and every case depended upon our securing these Wassermann data. If the Wassermann data were dubious, then a longer period of diagnosis was necessary. If the serum proved negative but there were other signs of organic disease of the nervous system, lumbar puncture was resorted to, with the diagnostic hope that the Wassermann reaction and the other reactions of the fluid would prove decisive. The practical disposition of each case I found to depend upon taking the Wassermann reaction of the serum. Accordingly, although there is no indicator symptom which is infallible or anywhere near infallible in the detection of neurosyphilis, the value of the Wassermann reaction of the serum is such that I feel entitled to place the syphilopsychoses, or mental diseases of syphilitic origin, at the head of all diagnostic consideration. This was the beginning of the plan of ordinal exclusion, the plan of diagnosis *per exclusionem in ordine*. The first thing to be excluded in the diagnosis of mental disease was, in short, syphilis.

The second thing to be excluded was, I found in practice, some form of feeble-mindedness. To be sure, the obvious imbeciles and idiots may secure their diagnosis by inspection, and not so much difficulty is ordinarily felt amongst the morons or the feeble-minded proper of the English classification. But when one came to the so-called stupids or subnormals, that we now find to lie between the morons below and the normals above, the value of the Binet-Simon tests or their analogues became extreme. One does not need to claim one hundred per cent values for these tests any more than for many another biological test. The tests are nevertheless of value. I found that in practice I needed to know of every lucid case, that is, of every case which could practically be tested metrically, the intellectual titer. Even were I dealing with such a condition as dementia precox or manic-depressive psychosis, I nevertheless found that I needed the mental test as a base line.

Thirdly, I found that the hypothesis of epilepsy or epileptic symptoms and of epileptoid phenomena is one very apt to be isolated from consideration by the tyro or even by the expert. I placed epilepsy third in the diagnostic order of exclusion partly because of the relative reliability of the clinical histories in these cases.

After Group I, the syphilopsychoses, Group II, the hypophrenoses (feeblemindednesses), Group III, the epileptoses, Group IV, the pharmacopsychoses (including mental diseases due to alcohol, drugs and poisons), I come to Group V, the group of mental diseases due to focal brain disease,—the so-called encephalopsychoses. This is the group of cases in which we unconditionally need the data of the routine neurologist's diagnosis. We need to exclude conditions of heightened intracranial pressure and conditions of asymmetry, reflexes and the like, indicating focal brain disease.

Sixthly, after Group V, the encephalopsychoses or neurologist's group, I found that in practice I was asking for the data of the internes, namely, the data which would go to make a diagnosis of a mental disease somewhere in the "symptomatic" group,—Group VI, a group I have termed the somatopsychoses.

After these I tried to exclude presenile and senile phenomena belonging in a group which I have termed, following Nascher's Geriatrics, the geriopsychoses (Group VII).

Then come the stock difficulties of diagnosis for the psychiatrist,—the diagnosis of Group VIII, the dementia precox group, which I like to term (from their main symptom as defined by Bleuler) the schizophrenoses.

After the schizophrenoses stand Group IX, the cyclothymoses, or a group including the manic-depressive psychoses and similar mental diseases.

The last of the ten well-defined groups is the familiar group (X) of the psychoneuroses including the hysterias, neurasthenias and psychasthenias; but if in the diagnostic order of exclusion one could not place one's case in any one of these ten comparatively well-defined groups, one would not throw it altogether out of the psychiatric lists, but would call it a plain psychopathia and put it in Group XI, the psychopathoses. A table of these major groups is shown on the following page.

I have spoken of the botanical and zoological analogues of this grouping in a paper read before the Neurological Association in 1917. Two words of warning are necessary concerning this analogy. The choice of the ending *osis* is obviously an attempt to get a medical analogue for the botanical ending *-aceae* or *-osae*, as in the orders *Rosaceae* and *Leguminosae*. These botanical orders are, as is well known, susceptible of splitting into genera and species. It would be an entirely superficial objection, for this account, to say that the term "species" has obtained in natural history a very special meaning which could not appropriately be given to a disease. Biologically speaking, a species is sometimes taken to mean such a group of plants or animals as are capable of breeding true to type. However, the genus-species distinction is a great deal older than natural history and is used throughout every province of logical

classification known. For example, the mineral kingdom, though without process of breeding, is in part susceptible to a classification of the genus-species sort. For that matter, it is not necessary to use the terms order, genus and species for these groupings at all. One might use entirely other distinctions, and still the process of classification might be entirely valid.

The other point which should be made is a kindred one. In zoology and botany one would ordinarily be satisfied if in the process of logical exclusion one arrived at the animal or plant which fulfilled the conditions of differentiation, but in the process of diagnosis *per exclusionem in ordine* as applied to mental disease one unconditionally needs to go through the entire list in each case.

For example, one might easily face an instance of mental disease such that the victim was a syphilitic, feeble-minded, epileptic, alcoholic old man, with focal brain disease and somatic disease, the whole contributing to his mental picture. He might conceivably have been psychoneurotic or cyclothymic in

Mental Disease Groups (Orders)

1. Syphilitic	Syphilopsychoses
2. Feeble-minded	Hypophrenoses
3. Epileptic	Epileptoses
4. Alcoholic, drug, poison	Pharmacopsychoses
5. Focal brain ("organic," arteriosclerotic)	Encephalopsychoses
6. Bodily disease ("symptomatic")	Somatopsychoses
7. Senescent, senile	Geriopsychoses
8. Dementia precox, paraphrenic	Schizophrenoses
9. Manic-depressive, cyclothymic	Cyclothymoses
10. Hysteric, psycho-, neurasthenic	Psychoneuroses
11. Psychopathic, paranoiac, et al	Psychopathoses

his life, for these conditions are doubtless consistent with a fundamental feeble-mindedness in some cases. Whether he could also have been a victim of schizophrenia (dementia precox) and still have mental phenomena attributable to all these other groups is a question. We are aware of cases that start by being feeble-minded and apparently have their schizophrenic process grafted upon them; but whether these combinations of psychoses in so multiplex a form are common or really exist is not my point. Combinations of two or three of these conditions are not infrequent. It is, therefore, stringently necessary, in the rough analysis of psychiatric cases, to make all of these major hypotheses and dismiss them or fix upon them one by one. In short, diagnosis *per exclusionem in ordine* does not necessarily exclude ten out of the eleven groups, but it does endeavor to exclude each one of at least ten of the groups. If the process has succeeded in excluding the first ten, the case, if psychiatric at all, must remain in the unresolved or ragbag group of psychopathias, Group XI, the psychopathoses. I do not intend here to enter the minutiae of psychiatric diagnosis, although in another place I have made some endeavor to describe the main genera and species of the groups above tabulated.[26] Incidentally, I have endeavored to place genera and species in the same sort of practical order, so that the tyro may consider the diagnosis

of the species of syphilitic, hypophrenic and other forms of disease in an arbitrary order.

It is scarcely necessary for me to insist that I do not attach great consequence to there being ten or eleven more or less major groups of mental disease. In fact, I insist that the future must interpolate or extrapolate these groups. The groups are not groups based upon clinical resemblances alone, upon anatomical disease, or upon etiology. They are practical groups devised upon what has been found to be the best practical order of exclusion of major groups of mental disease, each seeming to require a special form of handling. I have described the method in my paper entitled "A Key to the Practical Grouping of Mental Diseases." I am not sure that I would not be warranted in using the term pragmatic instead of practical, for the end and aim of these groupings is, after all, a therapeutic one. The basis for these rough distinctions is a question of disposal of the patient. If the patient belongs amongst the syphilopsychoses, then antisyphilitic therapy is indicated, or at least must be thought of. If the patient is hypophrenic, then a school for the feeble-minded, an institutional equivalent, or some form of supervision looking to the safeguarding of the community and the development of the case must be adopted. If the case is epileptic, certain special safeguards are necessary, whether the case is an alcoholic epileptic or a traumatic epileptic. The etiology and the anatomy are not at all so much to the point as the practical handling of the epileptic. So, too, with the alcohol and drug cases. In fact, the practical consequences of placing one's patient in one of the first four groups of syphilitic, hypophrenic, epileptic and alcohol or drug cases are such that I think on these practical grounds alone it would be wise for the diagnostician to consider syphilis, feeble-mindedness, epilepsy and alcohol, drugs and poisons before he comes to the minutiae. He will thus get settled in his mind many of the practical social questions of a legal, institutional and social service nature. Take the group of mental diseases due to focal brain disease. Should the phenomena of the present war show us that the traumatic psychoses, that is, psychoses due to structural brain disorder, form a practical group on a level of practical interest equal to that of the listed groups, I for one should have no objection to raising these traumatic psychoses, which I at present place under the encephalopsychoses, to a higher dignity, namely, that of the traumatopsychoses. For the moment, I seem to feel, and I suppose most of my colleagues would agree with me, that the traumatic psychoses rather resemble genera under the focal brain disease group than the true great orders of disease. Let the list be telescoped or accordeonized as you will, no damage is done to the concept-value of the greater groups of mental disease.

Again, should someone wish to consider the somatopsychoses ahead of the focal brain disease group and exclude e. g. pellagra and cardiorenal disease from a role in the given patient ahead of brain abscess, brain tumor, arteriosclerosis, and the like, I should have no objection. The point I make is that some order, not any particular order, is desirable for the tyro in diagnosis. I insist, therefore, on no particular great groups, but on the principle that

there should be great pragmatic groups of mental disease analogous to the orders of botany. I insist, again, on no particular order of exclusion, but on some order of exclusion of these groups, and above all, I insist upon subjecting each mental case, so long as indicator symptoms are so woefully absent, to the hypothesis of his possibly belonging under every one of these great groups. Indeed, I feel that he may belong in two or more of these groups.

Whither, you may ask, does the pragmatic principle then disappear? Suppose a man is both epileptic and alcoholic; shall we classify him as in the group of epileptoses or in the group of the pharmacopsychoses? All specialists will remember acrimonious discussions of this nature. Are we dealing with epilepsy or with alcoholism, or with both? Now, I find that in practice, if you couch the question pragmatically, there is rarely, if ever, any doubt into what group you wish to throw your case. Once you put the question thus: Do you wish to send this case to an institution for epileptics or do you wish to put him in an institution for alcoholics or under appropriate supervision suitable for alcoholics? There is almost never any doubt on the part of the careful physician whether his case is a case for an institution for epileptics or a case for an institution for alcoholics. The pragmatic question is forthwith settled. The academic question, whether one is an epileptic alcoholic or an alcoholic epileptic, becomes a vividly practical question when the pragmatic, that is to say, the therapeutic, question is raised. I find that this kind of consideration matures the tyro in psychiatry very fast.

SUMMARY

1. The writer apologizes for a communication on Medical Logic in general when he is only a psychiatrist and but recently a pathologist. His excuse is the necessity for reasonably accurate snap diagnosis in the sifting problem of the psychoses, psychoneuroses and psychopathias, as they flow through the Psychopathic Hospital clinic in Boston.

2. The medical student is found destitute of the ability to define entities and symptoms. The textbooks in medicine, especially the single volume textbooks, rather tend to make the student believe that diagnosis is observation. In point of fact, diagnosis is not observation, though it requires and indeed stands or falls by accurate observation.

3. Da Costa and his successors have lauded so-called direct diagnosis to the skies, and Da Costa rather decried indirect diagnosis by exclusion as a tedious process. An example is cited from Da Costa which shows how relatively simple the classical diagnoses of general medicine are beside those of psychiatry.

4. It seemed that diagnosis by exclusion ought to be rehabilitated. An examination of recent research work in logic indicated that higher and more complex methods than those of observation had become necessary in science. For example, Cabot and Herbert French have attempted to profit by the statistical method, which again, though it requires reasonably accurate observation, is not in itself a method of observation at all. Yet Cabot's statistical fre-

quency tables possess a certain diagnostic value. But the student is often misled by the brilliancies of so-called observational diagnosis in a clinic. Here diagnoses are often rendered on inspection by a process akin to the recognition of a fruit as an orange, or an automobile trouble as "the engine is skipping." This process is not diagnosis, it is a process of recognition that may receive a simpler term *gnosis*.

5. The offhand snap diagnostic work at the Psychopathic Hospital indicated that we were in practice relying upon the successive exclusion of certain great disease groups in a certain definite order.

6. A study of Royce's "Summary of Recent Researches in Logic" shows how an organized combination of theory and experience is the higher logic to which the more complicated sciences must resort. Royce himself mentioned psychiatry as a science about to climb out of the classifying era into the era of logical order, that is, of the organized combination of theory and experience. Such a dictum as that "disease is life under altered conditions" seems now childishly simple. The idea that disease is a matter of an organism plus a germ was found to be altogether too simple when in the nineties of the last century the concepts of immunology were developed.

7. Those departments of medicine in which the presenting symptom of Richard Cabot is of value are lucky departments. Those departments of medicine in which the indices of disease, or indicator symptoms of the elder writers are available, are also fortunate departments in comparison with psychiatry. In mental disease there are exceedingly few indicator symptoms.

8. Hence the need became apparent of a process of exclusion of great groups or phenomena in a certain definite order, so that nothing of large significance should evade consideration. To avoid the tediousness of exclusion, complained of by Da Costa, the phenomena of disease had to be logically grouped in certain great groups, and the process types of diagnosis in the books may be counted as six or eight, according to definition.

9. In the body of the paper, a special statement was made about each of these process types: Inspection (regarded as not really diagnosis but as merely recognition of gnosis, Expectation, a newly named but frequent method (far older than Micawber), Induction, *Ex juvantibus, Ex nocentibus* (three methods in which in no very rigorous way experiment is used) and three methods of diagnosis by comparison are successively discussed.

10. The ninth method, diagnosis *per exclusionem in ordine*, is in one sense a minor modification of the old method of diagnosis by exclusion. It is of value in departments of medicine where there are no indicator symptoms, and where the so-called presenting symptom would merely indicate some kind of mental disease.

11. The general application of the method of diagnosis *per exclusionem in ordine* in the field of mental disease is demonstrated in the eleven groups of mental diseases into which most of the phenomena may be pragmatically cast. The groupings are not by clinical resemblances, by anatomical attack or by etiology. The distinction is a pragmatic and therapeutic one and will naturally

tend to become more and more etiologic as the causes are determined. But in the field of mental disease, causes are so apt either to be unknown or to be multiple that etiologic classification on any simple basis such as that of the infectious diseases is practically inconceivable.

12. It is hoped that other departments of medicine (where diagnosis is raised above the level of mere recognition) and where there are few or no pathognomonic or indicator symptoms, will find it to their advantage to set up a method of diagnosis *per exclusionem in ordine*, the great groups or orders being always determined on a pragmatic basis. In the body of the paper are given the general designations of the ten great groups of mental diseases, with the eleventh residual group.

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LVIII

GENERAL PSYCHOPATHOLOGY

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The reviews and summaries of this number of the BULLETIN have always in the past three years dealt with general or theoretical psychopathology, familiar to us in the portions of psychiatric books termed "general," for example, in that portion of Kraepelin's well-known textbook termed "Phenomena of Insanity."

The present number has been delayed through various contingencies, incidental to the war. I have determined to make this fourth year of reviews and summaries a consideration of Shell-shock, leaving for another year such accumulations of general psychopathological literature as may be available. I wish to consider Shell-shock not so much from the medical as from the psychological side. In the preparation of a case-history book entitled *Shell-Shock and other Neuropsychiatric Problems Presented in 589 Cases from the War Literature, 1914–18* I spent about 2,500 hours, and my colleague, Mr. Norman Fenton, as many more in the preparation of the bibliography. The resulting dictations and bibliography constitute the major portion of this book of approximately a thousand pages, which I am not here endeavoring to abstract so much as to consider from the special psychological point of view. Dr. Charles K. Mills in a long introduction to this Shell-shock book has analyzed it from the general medical standpoint.

My own purpose in compiling the book was not a psychological one primarily. The object was to produce a case collection after

the manner of the law case books, which should serve the neuropsychiatrists in their preparation for war work. In fact, the task was undertaken incidentally in my work as director of the Army Neuropsychiatric Training School (Boston unit), 1917–18.

It has always been the task of these reviews of general psychopathology to take out from the mass of the medical literature that which promised to be of most interest to the psychologist. Of course the physician working in this field has a weather eye to the interests of the premedical students, with whom the psychologists in their college work come in contact. I believe that the psychologists and the psychiatrists ought to come very close together in this matter, since, if the students in their premedical work do not get the right slant and encouragement, the task of recruiting psychiatrists for the new work of mental hygiene will be difficult or impossible. About three per cent. of the physicians in the Surgeon-General's Office are said to have been neuropsychiatrists or at least posed as neuropsychiatrists. Psychologists coming in contact with young men having this sort of interest ought to encourage them definitely to go into medicine and particularly to attend those medical schools where proper attention is paid to psychiatry. A definite propaganda to this end ought to be launched, possibly by the newly established Education Committee of the National Committee for Mental Hygiene (this Education Committee has amongst its members psychologists as well as physicians). But, if the psychiatrists wish to recruit their own ranks, they must strive to put their new ideas in such form as rapidly to get into the minds of the psychologists. It is on this account that I have chosen to write a sort of review of my own compilation. Essentially, however, it is not a review of anything I myself have written so much as a culling out of the articles in the medical literature which seemed to me, on rereading my own compilation, to be of especial interest to psychologists. There are, in the bibliography of this work, articles as follows: French, 895; British, 396; Italian, 77; Russian, 100; American, 253; Spanish, 5; Dutch, 5; Scandinavian, 5; and Austrian and German, 476. The bibliography is much more extensive than the articles from which the abstracted cases are drawn. The bibliography has been brought up to and partially includes 1919. The matters of psychological interest in these two thousand references can be got out by author in the bibliography itself and by subject in the index of the work under the heading *Bib.*

The whole work is divided into five sections, of which the fifth is an Epicrisis that contains practically every thing that the compiler himself has to say (and that very little) concerning the general nature of Shell-shock, general observations upon its treatment, and various problems of diagnosis, which latter need not concern the psychologist as such. In the body of the work, Section A deals with the psychoses incidental in war, that is, with psychoses not necessarily related with Shell-shock or very doubtfully related thereto. Thus, to give a few instances of these circumambient difficulties, one finds in the literature cases in which general paresis or kindred syphilitic disorders of the nervous system have been brought out by shell shock or, better, following shell explosion. Again, genuine epilepsy has been thus brought out. Again, we find various diseases of the body at large, even such diseases as tetanus and malaria, presenting phenomena that may at first blush be confused with the true Shell-shock. It is rather surprising that the disease which so fills our asylums, namely, dementia precox, is not especially well represented in the war literature. It is doubtful whether the phenomena of dementia precox have been brought out by Shell-shock or by any special war influences (all this aside from the fact that numerous cases of dementia precox come up for diagnosis both in the field, in camp, and in the draft or enlistment stages). It is particularly striking that so few cases of depression of the manic-depressive group are brought out by war stress.

The second section of the book (*B*) deals with the nature and causes of Shell-shock and begins with matters of medical interest, namely, with autopsied cases and with cases showing signs of organic disorder of the nervous system. The psychologist here may inquire what, after all, physicians feel concerning the organic nature of Shell-shock. The answer must be that, statistically speaking, the majority of cases of so-called Shell-shock are no doubt *functional* (whatever that word may mean) in exactly the same sense as the hysterias and other psychoneuroses made familiar to us by the work of Charcot, Weir Mitchell, Janet, Freud, and others. In short, although minute brain hemorrhages are no doubt found in certain cases of Shell-shock, the majority of the phenomena are of that reversible nature to which we give the term functional. Shell-shock, to put it briefly, is a traumatic neurosis, not a traumatic defect-psychosis. Perhaps the warning does not need to be made that this truth is a statistical truth and that cases are met

in which there is a combination of the functional with the organic element. Papers by Babinski and by Binswanger deal with many of these combinations of hysterical and more narrowly somatic disorders.

The combination of French and German authorship noted in the previous sentence may give point to the remark that the general results on both sides of the battle line were practically identical. For example, Nonne, the well-known neurosyphilographer of Hamburg, found himself in the war treating functional cases by functional methods, preferably in the case of this eminent neurologist by means of hypnotism, to which procedure he appears to have been converted during the war. But, aside from details of treatment, it is remarkable that Nonne should have proclaimed during the war that the results of war studies lent more support to the original contentions of Charcot concerning hysteria than to any other authority. It may be remembered how the original contentions of Charcot were by some regarded as those of a charlatan, simply because he was dealing with new material with which the ordinary practitioners had little contact. The result of the war work will be that a knowledge of hysteria and other psychoneuroses will become very much more widespread. There will be three or four men in the future familiar with the psychoneuroses to one man in the past. All of which augurs a far more brilliant immediate future for mental hygiene than one could have hoped.

If the psychologist asks whether we really know anything more about the fundaments of hysteria and the other psychoneuroses than we did before, perhaps the answer should be no. Many authors have made the obvious point that at least the sexual portion of the Freudian doctrine is not upheld by the war experiences. Those who held to *fear* rather than *sex* as the more frequent cause of functional neuroses may regard themselves as supported by the war evidence. But, as is well known, the majority of the so-called Freudians are no longer pansexualistic. They are much wiser in clinging to the virtues of symbolism and to the values of dream analysis than to the doubtful universalities of the doctrine of pansexualism. There are a few cases in the compilation which are of interest from the psychoanalytic point of view, notably some cases from amongst those described by Eder in his book *War Shock*. Take, for example, case 359 of the compilation called (by the compiler) *A Horse in the Unconscious*. Or take case 529 *A Victoria Cross winner: Bayonet clutch contracture revealed by hypnosis*.

MacCurdy has also a number of cases well analyzed from the general standpoint of the Freudians, and reference should be made to his book which should be in every psychological library. Another author with psychoanalytic leanings is Rows. See especially cases 342 and 343 dealing with certain dreams. On the whole, however, as above mentioned, at least the pansexualistic part of the Freudian doctrine must be regarded as not well supported. A moderate statement in this direction is that of Elliot Smith and T. H. Pear in their book *Shell-shock*.

Many Freudian authors insist, however, that Freudian "mechanisms" are at work nevertheless and despite the lack of war evidence for the sex factor. Perhaps the compiler is stupid or prejudiced, but he has never been able to get clearly into his mind exactly what the term "mechanism" means as used by the Freudians. He has repeatedly replaced the term "mechanism" with the term "process" in writings of the Freudians, finding the resultant statements at times perfectly true, but far more obvious and everyday-seeming than when the aristocratic word "mechanism" is used. Section XI, the group of psychopathoses in the first division (*A*) of the work, may be referred to for cases to illustrate these "mechanisms." There is one most remarkable German case (Steiner) of a man who preferred going over a badly shelled area to a perfectly safe tunnel which had been provided for him, simply because he had developed a severe claustrophobia as the result of Shell-shock (Case 182).

Whereas the second section of the book deals with the general nature of Shell-shock, the third section deals with more special problems of diagnosis which do not especially concern the psychologist, save in the work of Babinski on the relation of certain reflexes to chloroform anesthesia, a matter which will be considered below more extensively. The fourth section of the book (*D*) deals with treatment, and this section is in some respects the most interesting to the lay reader and the professional reader interested in reconstruction. With this brief account of the general construction of the compilation, I will return to a more detailed consideration of that material which seems to me to be of the most psychological value.

Concerning feeblemindedness, the cases presented in the group 35 to 52 of the psychoses incidental in the war must give a good deal of concern to the psychologist. Even when the psychiatrist is persuaded concerning feeblemindedness, the regimental surgeon may fail to agree with him (Case 34). How could a good rifleman

be an imbecile, was a question raised (Case 45) by a certain German. Sundry superbrave imbeciles and imbeciles fit for barracks work, although decidedly unfit for war work of a more active nature, are given. One imbecile who stood his ground as a model of the brave soldier was finally captured by the Germans still shooting amidst a hail of bullets. However, nothing daunted, he escaped from his captors and swam back to the French lines across the Meuse (Case 36). There are cases in the compilation of the greatest interest from the standpoint of "rationalization." Case 51 seems to be a case of Shell-shock in a feebleminded person following burial in an explosion. The victim was thus complicated enough to secure a Shell-shock in the sense of a functional neurosis, but hardly complex enough to rationalize his situation properly. As one might say, such a man is like a cat able to climb a tall tree, but unable to climb down—whereupon the entire fire-department of the cat's native village may be called out. As for the interesting process of "rationalization," the excellent work of the well-known psychologist Rivers may be quoted. Rivers' original work should be read carefully by the psychologist. Cases 506–510 are instances of the rationalization process as applied by Rivers. Case 510 is one in which there was no redeeming feature whatever, because Rivers could find no nucleus of rationalization on account of olfactory, gustatory, visual, and auditory elements that drove in the neurosis. But ordinarily Rivers was able to find some feature, however slight, in the neurosis-producing situation to which the mind of the victim could be attracted. Thus, if one's comrade had been blown to small bits by one's side, then the rationalizing point lodged in the very fact of the rapidity of his death: he could not have suffered at all, and that was at least one point of advantage. This philosophy of rationalization is of course entirely opposed to the ordinary "forget it" philosophy of some of the psychotherapists, notably of the Christian Science group.

One gets the impression, however, that these processes of rationalization of the psychoneuroses are far more fit for officers than for men. The general conclusion seems to be that these more elaborate neuroses are in the nature of the case more likely to occur in more complicated human beings and that by the same token the more complicated methods of treatment must be reserved for the more civilized patients. The part played by subnormality and even by moronity in all wars must now be regarded as an extensive one and the fact that a great quantity of these cases can be eli-

minated by simple psychometric methods must give the world a considerable ground for optimism in the future. Even should there be no more wars, these methods can well be applied to industry.

Where we are not dealing with feeblemindedness, what must be regarded as the basis of Shell-shock? Hysteria used to be regarded as almost always hereditary. A great many cases with heredity have been found in the war time. Yet excellent neurologists are found to assert that they have had typical and well marked cases of Shell-shock under observation, whose histories have been studied elaborately and no hereditary or acquired psychopathic tendency has been found. Thus, a case (306) of Donath was published by the author with his explicit statement that here was a case of traumatic hysteria without heredity or acquired soil, and MacCurdy has a case (307) of mine explosion and burial with neurosis ensuing in a man regarded as a perfectly normal and very high type of soldier. On the whole, however, it must be said that war conditions are not very suitable to proper social service investigations, and eugenic investigations into the heredities of these cases in the majority of cases do show either hereditary taint or acquired soil.

Of great general psychological interest appeared to the compiler the great number of cases in which there had been *ante bellum* difficulties of the same sort as those shown in the war. Thus, a man whose leg was paralyzed after falling from a horse under fire was the same man who had had precisely the same disease after a fall from a horse in a sporting adventure years before (case 286 of Forsyth). The lamented Dejerine has shown that a subject who had always been weak in the legs developed especially marked weakness in the legs under the war conditions. There is one very striking case in which a soldier with hysterical chorea was found to have had a precisely similar attack years before the war: this *ante bellum* attack was reminiscent of a chorea in the man's mother, but the mother's chorea was an *organic* one (case of Dupouy 300). It would seem that there might be developed a general theory concerning weak places in the body to which symptoms might get attached. One of the most productive neurologists in the war, Tinel, has a case in which tremblings of the eyeball developed along with sundry other symptoms in a man who had been waked by the explosion of a shell. According to Tinel, this nystagmiform tremor was an "occupational reminiscence" in a cinema worker (case 315).

Of course the well-known logical situation is repeatedly found, namely, that there can be Shell-shock *without either shells or shocks*. In short, there may be diseases looking like Shell-shock but related with no acute phenomenon whatever. Thus, Wiltshire speaks of a man who heard a shell explosion, but did not develop symptoms until he had heard distant artillery twelve days later. Lattes and Goria have a case (322) of a man who was jostled while he was carrying some explosives. He did not drop the nitroglycerine, but nevertheless fell into the state of unconsciousness and deaf mutism with later the so-called "*camptocormia*," a special rather new kind of hysterical "bent back" developed in this war. One Frenchman acquired the *croix de guerre* and his Shell-shock simultaneously. This man was another of those with "reminiscent" phenomena. He had a hallucinatory bell ringing which reminded him of the ringing of the bells outside of a Parisian moving picture show (case 314). These doctrines of the preëxistent "weak spot" and of the relation of certain Shell-shock phenomena to *ante bellum* experiences will no doubt give rise to many hypotheses concerning so-called "mechanisms."

The cases in the part of the book that deals with the nature and causes of Shell-shock are arranged for medical purposes from below upwards. Thus, the cases involving one foot or leg are followed by the cases involving both feet or legs, then by cases affecting one hand or arm, then by both hands or arms, and finally there is a sheaf of cases dealing with symptoms more related to the head. We here deal with mutism, deafness, blindness, and the various disorders of memory and personality. These latter cases, beginning perhaps with case 318 and running to case 370, are of special interest to the psychologist. These cases deal with the relation of emotion without shell explosion to the development of symptoms. The matter of tremors, of dreams, loses of consciousness, stupors, comes under consideration. There are three interesting cases of Milian (364-366) of somnambulism of many days duration (one case of twenty-seven days duration) with cure following a minor suggestion. Case 369 of Feiling is one of dissociation of personality. Beautiful war dreams may be developed by a man who has never seen any war service at all and has been hundreds of miles behind the battle line (Russian case of Gerver, 347).

Behaviorists ought to be especially interested in another case of Tinel, a case of what he calls "stupefaction" of a muscle (case 253). The biceps had here been contused and became as it were

stupefied, while the supinator longus still functioned. The following case (264) of Tubby also relates to the blockage of impulses to certain movements of the arm, and cases of the psychologist Myers are particularly to the point in the analysis of inhibition. That theorist who shall go very profoundly into the nature of Shell-shock will have to reckon not only with the "weak spot" and "*ante bellum* trend" hypotheses, but he must take account of the fact that the symptoms are so often on the same side as the explosion. In some cases it would seem as if the muscles on the side of the body where the explosion occurred were paralyzed (and the overlying skin rendered anesthetic), whereas the muscles on the other side of the body were thrown into contraction,—almost as if the part opposite to the explosion was trying to run away therefrom, while the parts near to the explosion were transfixed upon the spot. The behaviorist must gain a great deal to his purpose from this group of cases with asymmetrical symptoms on the two sides of the body. Especial attention should be given to the work of Babinski. Whatever the truth of his contention that the so-called reflex disorders are incurable by suggestion (other authors, notably Roussy, seem to have been able to cure by suggestion certain cases that Babinski calls "reflex"), nevertheless, the theoretical contribution of Babinski upon the nature of these so-called reflex disorders must certainly be conceded. According to Babinski, these ideas simply conform to points made years ago by Charcot and Vulpian, but neglected by later workers. Babinski's main point is that in certain stages of chloroform anesthesia unsuspected conditions of the nervous system can be brought out. Whereas it has been thought that anesthesia ought in general to reduce the reflexes and whereas this is in general true, nevertheless there is a phase whilst going under and coming out of chloroform anesthesia in which the reflexes may come out in excess. Let us suppose a patient whose knee jerks are perfectly equal in the waking life; let him be chloroformed, and one of the knee jerks early in the anesthetization becomes very much exaggerated or even polykinetic. How is this to be explained? No doubt, the anesthesia has removed the normal downstream of inhibitory influences which physiologists for many years have attributed to the brain. In short, Babinski by chloroform anesthesia is producing an effect not in any wise logically different from the exaggerated knee jerks produced after cutting through the spinal cord. In both instances the downstream of inhibitory influences from the cerebrum has been cut off. In this

way Babinski feels that he has shown the existence of functional differences on the two sides of the body which could not be demonstrated in normal life. The reviewer cannot here do justice to these contentions which ought to be read by the psychologist in the book by Babinski and Froment on *Hysteria*.

One of the most striking illustrations of the Babinski theory is to be found in an article by Monier-Vinard (see case 280). Monier-Vinard had to do with certain cases of tetanus, the victims of which had apparently entirely recovered after a period of some weeks. For certain reasons, it became desirable to operate upon these men for orthopedic defects. To the astonishment of the observer, under chloroform these men redeveloped tetanus and showed a degree of rigidity in anesthesia which was highly alarming. Upon removal of chloroform these rigidities disappeared, only to reappear upon further chloroforming. The only hypothesis ready to hand is that although these cases were clinically cured of their tetanus, nevertheless there was within their nervous systems a tendency to hypertonus. This tendency to hypertonus was counteracted, no doubt, by the normal downstream of inhibitory influences from the cerebrum, and it was this normal downstream that had been interfered with by the chloroform anesthesia. Here then, we have laid down for us the basis of an ingenious hypothesis concerning concealed functional disorders. Suppose we apply this hypothesis to the cerebral cortex itself: we can well get an image of what may be the basis of, let us say, so delicate a disease as the fixed idea. Far be it from the compiler to insist that this is the true account of the basis of a fixed idea or of any similar notion in the psychopathic field. It appears, however, as if a new weapon was in the hands of the psychopathologist. Let us suppose alcohol to work upon a man with certain inhibited tendencies (tendencies which Freudians might like to call repressed), the alcohol might work after the manner of the chloroform anesthesia in the Babinski cases, and the special tendency be released precisely like the exaggerated knee jerk under chloroform.

A word remains to be said concerning treatment. There is no "one best way" for the Shell-shock group. The compiler roughly threw the cases into three groups; a group of spontaneous cures, that is cures without medical credit attached thereto; secondly, a group of what might be termed "miracle cures";and thirdly, the reëducative group of cures. The miracle cures are of several groups. According to Bernheim a suggestion is an *idea*

accepted. Although this definition is no doubt too broad, yet it has its advantage. Sometimes the suggestion is accepted through the means of pseudo-operations, such as the cure of blindness by an injection of salt solution in the temple or the cure of mutism by manipulations of the laryngoscopic mirror. Again, the suggestion is best put in by means of hypnosis, and a considerable group of such cases has been abstracted in the compilation. It appears that the French army authorities did not favor the use of hypnosis and that there were regulations enforced against it, at least in some parts of the army. But certain Englishmen and certain Germans used the method with great success. Probably the best known method is the so-called "psychoelectric" method, used to such advantage by Vincent in France, by Yealland in England, and by Kaufmann in Germany. This somewhat brutal method of treatment proved none the less successful, though one victim cured thereby carried his case high up amongst the French authorities and caused a great deal of trouble to his deliverer. Some of the most picturesque accounts of this method are to be found in the book of Yealland called *Hysterical Disorders of Warfare.*

When all is said and done, however, many cases remain outstanding in which all methods have failed and this despite the claim of 100 per cent. results by sundry authors. In almost all instances the cases of 100 per cent. claimants turned out to have been selected. Accordingly, a great deal of scope remains for reconstruction work and for reëducative methods of a slower nature. Herein no great advance seems to have been registered over the work of Weir Mitchell. But I am here entering more narrowly medical fields and thereby transgressing the scope of this review, which has been intended to bring out the main things which the compilation showed of value to psychologists. I am bound to say, on looking over the compilation, that a good deal of similar compiling work might well be done in the *ante bellum* literature, for our psychopathological and psychological literature has become too full of general statements concerning one or other hypothesis and too little provided with the actual case material in hand. It is to be hoped that some such complete analysis of the previous literature may before long be made. If so, we shall have side by side the precisely identical results obtained by physicians, ecclesiasts, charlatans, and others, the whole situation depending perhaps upon the idea of suggestion. The problem of suggestion then in its true nature remains the big problem of psychopathology and psychology.

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LIX

THE RANGE OF THE GENERAL PRACTITIONER IN PSYCHIATRIC DIAGNOSIS *

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One cannot help marveling at the progress made by psychiatry in the American medical scene. But the standing of psychiatrists and the prominence of psychiatric topics among medical men at large is more than paralleled perhaps by the vogue of psychiatrists and psychiatry in the broader circles of mental hygiene. It has been my privilege, as delegate from the Commonwealth of Massachusetts, to attend in the last few years many meetings of nonmedical workers, such as meetings of social workers, psychologists, general scientific workers, and even of teachers of philosophy; and I can report that this associate medical world (if we may call it so) is ready to go more than half way in meeting physicians on a mental hygiene platform. Too eager to enter on unsuitable tasks are these nonmedical workers, some advocates of a Little Medicine might aver. But the Greater Medicine should open its arms to what Weir Mitchell called the "assistive" arts and sciences and, in matters touching the community, give over all that savors of a closed shop for M.D.'s only.

Though the face which psychiatry presents to the "assistive" nonmedical branches of mental hygiene is most respectable (the osmotic interchange perfect, one might say), the interior relations of psychiatry to the rest of medicine form something of a problem. One difficulty, which I do not here more than mention, is the proper adjustment of the planes of contact between clinical neurology and psychiatry. The

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research basis of these two divisions of practical medicine forms a continuous unit, having structural and functional aspects, to be sure, but nevertheless growing steadily together.

Leaving the interrelations of neurology and psychiatry to themselves for the moment, I come to my chosen topic. I consider that, all in all, the appreciation of psychiatry by the general practitioner has not kept pace with the increasing appreciation of it now being shown by neurologists and with the even greater advance in the esteem in which psychiatry is held by social workers, psychologists, philosophical and ethical teachers, and other practical and scientific types of nonmedical mental hygienists. In short, the general practitioner ought to enter consciously and confidently the field of mental hygiene, some of the tasks of which lie very near his hand.

But why is neuropsychiatry caviar to the general practitioner of medicine? It is also, we must admit, caviar to the general theorist, in American medicine at least. A determined effort to overcome the stepmotherliness of practitioners and theorists toward the general topics of this section of the American Medical Association must soon be made, if we are not to be quite outstripped by other nations even on the barest practical lines. I do not assign our unpopularity to inborn deficiencies of American practitioners. I am not even sure that our medical theorists are altogether responsible for averting their eyes. I am a little more inclined to fix the blame on the pedagogic group in our medical schools. Not that appropriate curriculums fail to be arranged, and not that clinical opportunities fail to be thrown open to medical students. But there is on the part of teachers in other branches than the neuropsychiatric an almost constant avowal of their lack of interest in nervous and mental diseases. This avowed apathy can hardly miss exerting its effect on the weary medical student, looking (as is his duty) for *things not to learn.* But what is the cause of the pedagogic look askance at nervous and mental diseases? Some of our own specialistic colleagues, who talk glibly of everything as a defense reaction, intimate darkly that neuropsychiatry is a sore subject with most medical teachers because they are so

ignorant thereof. Be that as it may, I feel that not all the blame attaches to our nonpsychiatric colleagues, and that our own obscurantism has blocked progress.

How much can be done with a frontal attack is doubtful. But correspondence following a paper presented to this section last year on a similar topic led me to think that a flank attack on medical teachers and research controllers might not be necessary to secure greater interest in mental hygiene on the part of the general practitioner. In the first place, the psychiatrist is, in a different way, almost as general in his approach as the general practitioner. The map quality and representativeness in the nervous system of virtually all other systems in the body insures a consideration of many things by the psychiatrist after the precise manner in which a general practitioner faces the same situation. This generality of view (e. g. that a psychiatrist, like a general practitioner, has to be something of an endocrinologist, a good deal of a syphilographer, etc.) is just as advantageous and just as dangerous as the generality of view which it is the duty of the general practitioner to have. Again, the psychiatrist, far more than any other specialist (unless perhaps the obstetrician), has to take his patient as a total person, as a globar individual—and this is precisely the duty of the general practitioner, and precisely the difficulty of the office consultant specialist as we usually see him. In passing, it may be noted that one of the reasons for the augmenting vogue of group practice by associated specialists is the demand for a more globar and comprehensive view of each patient. Nevertheless, it must be conceded that even group practice often leaves the patient a *dividual* (as old scholastics said) rather than an *individual,* and that the studies made are apt to remain on an analytic basis rather than aspire to the higher terrace of synthetic work. Otherwise put, the *dividual* is considered to have flat foot for the orthopedist, astigmatism for the ophthalmolgist, gastroptosis for the surgeon, and so on: the *dividual* patient remains a congeries rather than a collection of symptoms. This is a quarrel which the general practitioner and the psychiatrist lunge in common against specialized medicine.

Perhaps no more need here be said of the community of views and tasks between general practitioners of medicine and specialistic practitioners of psychiatry. A presupposition of my argument is that the general practitioner, if he is to live up to the designation "general," ought to grasp and use relatively as much in any one specialty (e. g. dermatology, ophthalmology, psychiatry) as in any other specialty. But, as the general practitioner is ready to agree, he certainly does not know as much about psychiatry as about many other specialties. How shall he proceed?

In the first place, there is often a bit of an obsession to rationalize. The general practitioner feels his education in psychiatry deficient and any postgraduate instruction practically impossible. He develops a general *psychopathophobia*. Moreover, if in his presence some psychiatrist inadvertently uses a term like *psychopathophobia,* it becomes easy for the old outcry about terminology to be raised. As a matter of fact, the terms of dermatology or of the chemistry of metabolism are just as difficult as those of psychiatry, often more so. What we are dealing with (to continue with this naive terminological joking) is another phobia, *onomatophobia*. One must, of course, acknowledge that terms for exact and complex things can hardly ever become much less precise, and much more simple than the facts, without destroying the facts. But there are signs that terminology is solidifying and getting less and less equivocal in psychiatry. In any event, the immediate duties of the general practitioner do not carry him into actual mares' nests of terminology.

Suppose the *psychopathophobia* and *onomatophobia* to be sufficiently rationalized. There is another comment on the relation of the general practitioner to psychiatry which has an optimistic tinge. Judging him by the standard of his range in other specialties than psychiatry, the general practitioner is found to be comparatively well up in major aspects of psychiatry.

I will not repeat in detail the arguments before presented concerning the general practitioner's reasonably sufficient equipment for handling the syphilitic, feebleminded, epileptic, alcoholic, focal brain (that is, these disorders in their major aspects), somatic ("symptomatic"), senile, and even perhaps the psychoneurotic

disorders. Inadequate practice, at any rate grossly inadequate practice, is not often found among these disorders, if we judge the practitioner by his achievements in other specialties.

In order to learn something of the status of the general practitioner with respect to psychiatry, I have looked over 500 brief descriptions of mental patients going to the psychopathic hospital in cases of the group we call the "temporary care group." That group is composed of patients who have never passed through the probate court or any judicial procedures. They are cases which physicians, or in some instances the chiefs of police and heads of boards of health, have thought suitable for observation at the psychopathic hospital. There is nothing mandatory about the hospital's receiving these patients for observation. The law says that the hospital *may* receive these cases for observation. Accordingly the admitting officers must make some preliminary observations of their own before the patients are admitted for observation. It must be carefully noted that these patients are *admitted,* not committed, to the hospital. A similar law to that for temporary care in Massachusetts is also in force in the municipality of New York. The law forms, as is well known, the acme of procedures in the interest of the mental hygiene of a community, as the modern psychopathic hospital acts as a magnet to bring psychopathic and all sorts of acute, curable, incipient or dubious cases to the state's observation and care.

For my present purpose, however, the point I wish to bring out is that this clinical material is very similar to and almost identical with material which, in most parts of the country, remains for many weeks or months under the control of general practitioners without recourse to the courts. Something like 1,200 or 1,500 patients of this order are under observation at the psychopathic hospital in Boston during a period of a year. They pass rather rapidly through a hospital of 110 beds, operating at from ninety to ninety-five beds, and are then passed back into the community or forward into receptacles for more chronic cases. Something like one third of all the patients are turned back into the community under proper supervision or,

if possible, without supervision. This material, then, is exactly what the general practitioner has to deal with, though in most states he has no recourse to a modern institution like a psychopathic hospital. Accordingly the remarks about these patients made by the general practitioners will probably indicate what the status of psychiatry is in the mind of the general practitioner in Massachusetts.

Of course, among these "temporary care cases" there will be a number which have passed through the hands of specializing neurologists or psychiatrists.

A report made by such a specializing neurologist may run as follows: "Emotional deterioration, apparent impairment of memory. Hallucinated, aural. Depressed. Delusions, somatic type. No initiative. Listless, apathetic. Poor cooperation. No capacity for work." Such a report is informatory, suggests the diagnosis dementia praecox, and is a fair sample of the routine report of a neurologist. The idea "emotional deterioration" is in our experience almost beyond the range of the general practitioner, nor have the words any very profound meaning for many excellent internists. Yet it would be comparatively easy in a brief period of observation in almost any hospital for the insane (not necessarily a psychopathic hospital) for a general practitioner or an internist who wished to brush up this specialty to learn the special meaning of a phrase like emotional deterioration.

Another neurologist's report speaks of an army case as "showing marked motor acceleration, flight of ideas, and press of activity." These are phrases due to the effect of the Kraepelinian psychiatry on our American institutions. I am not at all sure that I believe the general practitioner of medicine in America should learn all the slang of the psychiatric clinics; but the general ideas underlying the differential diagnosis between dementia praecox and manic depressive psychosis ought certainly to be understood by every general practitioner.

We also receive many cases referred from general hospitals, some of which are of the highest standard. The only instance in which catatonia was specified in any of these reports came from such a high grade general hospital. The fact that an assistant physician in a general hospital should know of the existence

of catatonia and be able to specify it in a report on a case ought not to be a remarkable fact. Still it is an isolated fact in the series of 500 cases whose temporary care data I looked over. We, I believe, did not determine that the catatonia in question really was a catatonia, but that is not the point I make. How many general practitioners are advised of the existence of catatonia and really envisage catatonia as they might sundry conditions in specialties like dermatology?

From a very good general hospital we received a statement something as follows: "Onset sudden with acute retention of paralysis (flaccid) of legs ten days ago. Severe pain in lumbar region preceding above. Temperature of 103 to 104. Remains high. Mentally deranged for about twelve hours." Mental derangement! This is a phrase like several others—"mental failure," "irrationality," "mind unbalanced," "cerebral degeneration," "nervousness," "patient erratic"—which phrases all seem to dodge the specific issue and to argue an utter lack of specification in the mind of the practitioner as to different forms of mental diseases and symptoms. There will be found in these reports excellent detailed accounts of the radiation of pain, the situation of tenderness, the Wassermann reaction, the phthalein test with digitalis, the appearance and disappearance of edema of the ankles, and the like; but there is no corresponding objectivity in the account of mental symptoms.

To our astonishment, we one day received from a high-grade general hospital the following: "Patient has been moderately depressed. Yesterday and last night patient became confused, showed fleeting hallucinations and ideas of a somatopsychic character, somewhat nihilistic. Unreasonable. At times disoriented. Difficulty in keeping the patient in bed or on the ward." We afterward discovered that this report had been made by one of our own former psychopathic hospital interns who had become connected with the staff of the general hospital in question. Here was a very informatory report, but one apparently quite beyond the range of most hospital practitioners.

Let us now come to the general practitioners themselves. There is a group of reports in which abso-

lutely nothing is said of value. For example: "A violent and dangerous maniac" *seems* to say a good deal about a patient, who, however, turns out to be neither maniacal, dangerous, nor violent. Another statement, "This man has been acting queerly for the last six months and seems to be all run down," is of little value, as also the report, "Has been gradually becoming mentally weak and physically weak." With all due allowance for the carelessness of some practitioners and the fact that many things may be said in conversation that cannot be put on official slips, it is clear that there is a good deal of dodging the issue, and what in the military service was so universally known as "passing the buck," in the field of general practice as it touches psychiatry.

On the other hand, there are numerous perfectly objective reports which, though they indicate absolute ignorance on the practitioner's part concerning psychiatry, nevertheless are very informatory, for example, "Crying and laughing. Breaking furniture. No sleep for three nights. Very destructive. Burned $150 in bills. Threw watch and chain away. Burned up new shoes and clothes." There is a report which tells something, though from other evidence we are aware that the physician in question has no claim to psychiatric knowledge.

Here is another objective report: "Patient does not sleep, is depressed, is afraid she is going insane, and has expressed a desire to die, but has never spoken of an attempt at suicide. The patient says she is worried about something in the past which she can never tell any one. Patient has an irregular heart, possibly on an arteriosclerotic basis."

Or take another objective report: "This man sits and broods, avoiding the company of other people always. He will not enter a street car if others are present. If empty, he will probably go in and sit down. He says other people have no use for him. He imitates actions of others in minute detail. He says he can't go on living this way."

These objective reports indicate clearly that we have in many practitioners a great deal to build on in our propaganda for the extension of psychiatric knowledge among general practitioners.

I have collected a number of instances of the grossly inaccurate use of phrases. Sometimes the users of these phrases are otherwise objective enough. Their inaccuracy merely points to their inadequate medical education. They are perhaps good observers, but poor reporters.

For instance, the statement that a girl has "hallucinations that a fellow has moved into the same house with her" means in modern technical phrase not "hallucinations" at all but "delusions." This confusion of hallucinations and delusions is the most common error in these reports. You may regard it as a venial error, seeing that as a rule the context will tell the psychiatric examiner whether the reporter meant delusions or hallucinations. That is of course true. But my point is that this confusion argues elementary ignorance on the part of the general practitioner, since practically the first thing which a medical student learns concerning psychiatry is this distinction between hallucination and delusion.

A more gross error in phrasing is the following: "Dementia resembling circular insanity developed." The fact that circular insanity, even if the phrase were now in common use, is a nondementing psychosis and does not resemble a dementia ought to be common knowledge among practitioners.

Such ignorance as this can readily be repaired by postgraduate work, and it is clear that a propaganda to this end will meet general approval.

It will be more difficult to eradicate odd ideas concerning etiology. In answer to the question, "What is the cause of the patient's mental derangement?" facts from the anamnesis are almost at random picked for exposition. Thus, "Patient gave her wrong age in securing a position and said that she had two brothers in the army. She feared she would be found out in these lies and arrested." Another, to the question, "What is the cause of the patient's mental derangement?" is *"in love?"* Another, "Overstudy." Another, "Has worked very hard for the past six months."

These facts on examination seem to be merely items in general situations. No one nowadays denies the psychogenic origin of a certain portion of

mental disease or the psychogenic factor in every mental disease, but we must certainly do something about our statistical blanks if we are not to perpetuate fantastic etiology in the common run of cases. A patient, who is described as having "fallen forty years ago and injured his back, excited, destructive, depressed," has as the cause of his derangement "high blood pressure." This patient turned out to be a case of hemorrhagic encephalitis. Another sample: "Patient comes in after an argument over something. In this case it was over the proper way to fix the bent stern of a submarine chaser which he was working on, and he being a house carpenter undertook to make the ship carpenter do it his way, hence the attack."

I need make no further comment on these etiologic statements. An extension of our propaganda to the demonstration of a few samples of the major groups of mental disease in clinics for general practitioners given at the various hospitals for the insane nearest the centers in which general practitioners might congregate would no doubt serve to erase most of the etiologic rubbish here hinted at. But the propaganda would also wipe out some of the grossly inaccurate use of phrases above mentioned.

Meantime the internists and general hospital physicians would have to brush up their psychiatry and undergo a reeducative or an educative process. All of which would minister to the mental hygiene of the community.

SUMMARY

Psychiatry has become almost more popular with nonmedical mental hygienists than the medical profession. Of course, the relations that are ultimately to stand between clinical neurology and psychiatry are not entirely clear. But the relations between psychiatry and the general practice of medicine are disturbed by special difficulties, e. g., phobias on the part of the general practitioner concerning nomenclature and concerning his own supposed ignorance of psychiatry.

A frontal attack is proposed on the general practitioner, in addition to the flank attacks considered desirable in the past, for his proper postgraduate education.

Psychiatry is more a synthetic art than is clinical neurology, now predominantly analytic. But, being synthetic, psychiatry has much in common with general medicine. General medicine, psychiatry, and (to a certain point) obstetrics treat the patient as an *individual,* whereas the majority of the specialties treat the patient (in scholastic phrase) as a *dividual.*

The body of the text contains material illustrative of some inadequacies of the general practitioner *re* psychiatry. Many of these are easily reparable.

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CROSS-SECTIONS OF MENTAL HYGIENE, 1844, 1869, 1894.*

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My task this day is definite and peculiarly impersonal. I must say words befitting an old and noble anniversary. Yet I must speak with far less knowledge of the turn of those years than could many amongst you.

I still contemplate with astonishment my election to the office of president in your association. I was deeply touched by the honor. Not a hospital superintendent, I had developed apparently from my cradle neurones and hormones of another sort, almost unfitting me for those indispensable and even essential tasks of most intricate generalship that are sustained by our hospital chiefs. Accordingly, I could in no sense represent the initial thoughts or inborn habitudes of the Original Thirteen or of their successors for many a year.

I might, to be sure, on second thought, somewhat better image the ideas and habitudes of the assistant physicians and other auxiliary officers who later entered the association. I am pleased to think, therefore, of my choice as a bit of an index of the remarkable and still rising democracy of our institutions, which has not been so much forced as fostered by the great advances in medical technique and the increasing complexity of total problems in the institutions which our association still so largely stands for.

Whilst we were assistant physicians in institutions for the mentally sick (as has been the experience of the vast majority of members here assembled), many of us must have thought of our superintendents as *despots*—benevolent despots, if you will, but still as despots. With the process of years and on augmenting

* Presidential Address at the seventy-fifth annual meeting of The American Medico-Psychological Association, Philadelphia, Pa., June 18-20, 1919.

knowledge of boards of trustees, boards of control, ways and means committees and other stimulating or inhibitory apparatuses, we youngsters tended to forget the aspect of despotism in our superintendents and to admire the benevolences which they somehow struck from the rocks.

Society emerges from barbarism, they say, through stages of *kinship* (in which the societal units are built up on the basis of families), of *authority* (where the despots and feudal powers come in), and of *democracy*. The development of professional institutions, such as those our association largely mirrors, has not been reduced to an exact history or approximate formulation. But I seem to see in the course of things a progress from (a) the simple institution where all were but one really almost happy family, to (b) the larger institution wherein all the fuss and strain of authority takes some time to adjust itself, and finally to (c) a situation with much division of medical labor and the development of specialized men and women (such as pathologists, clinical directors, roentgenologists, chemists, bacteriologists, psychologists, statisticians, social workers) often owing no intellectual allegiance to their superintendents. We have reached, in fact, times of great complexity in the tasks of psychiatry and of mental hygiene.

I venture to predict that before many decades, with the increase in specialized treatment, there will be established in institutions for the insane governing systems more like those in large general hospitals. In some of these latter, the superintendent, the physician-in-chief, the surgeon-in-chief, and even the pathologist, upon a basis of equality, form boards of government reporting direct to overseeing boards.

Far more complex and sometimes far more troublesome will be those new conditions. Justice Holmes somewhere remarks that civilization consists in the maintenance of complexity. This, I firmly believe. And I find in Holmes' principle the justification of democracy as against autocracy,—democracy is more complex and therefore, if it can be maintained, more civilized. Evil cannot exist in an elaborate system of checks and balances.

Enough said, then, of the at first sight strange contingency that a man not a hospital superintendent should have been chosen to my present office. Viewed aright, it is but a natural incident in an impersonal development of very broad tendencies.

I spoke just now of civilization. Civilization makes me think, of course, of my own Massachusetts! It is curious that 75 years ago the first president of this society, Dr. Samuel B. Woodward, hailed also from Massachusetts. We meet, as the Original Thirteen met, in Philadelphia, though not in Jones' Hotel, but in the Bellevue-Stratford. Again, too, our secretary to-day, Dr. H. W. Mitchell, is from a Pennsylvania institution, as was his predecessor, 75 years ago, Dr. Thomas S. Kirkbride.

The geography of personalities and of institutions—did not someone collect such ideas under the name anthropogeography?—is as interesting as their chronology. The predominance of New England and the Atlantic seaboard in the early years of our society reflected simply the greater advances in civilization there, with the casting up of more and more human by-products as objects of psychiatric study and the immediate discovery of men to make those studies. But I seem to see the fluid ideas of our association sweeping back and forth over the country as for the development of a vast photographic plate, and the presidents and other officers of the society, at first confined to Atlantic centers, move southward, westward, and northward with more and more even ictus, as the course of mental hygiene as a whole develops. Once we ventured as far as Colorado for a president, and Canada has had its due share of representation.

But to-day time must take our attention more than space. I believe that we can claim for our society the superlative in age for medical societies of national scope now in continuous existence in America, and I know that we shall maintain that Nestorian and no small honor far into the third millennium and, praise God, so long as the nation lasts.

The nation! I feel that as a society we should salute the nation (and I do not forget the Canadians and their correlative duty) year by year, and reaffirm our faith therein—the more so as no doubt the passage of years will confide great aspects of the trust of those years to us psychiatrists, the immediate guardians, not only of disease and defect, but also of the hygiene and the wholeness of the mind.

If we are the oldest surviving national medical society in point of mere years, we are as young as any in point of the spirit. Nay even, I hear that we may change our very name in keeping with

the passing phases of said spirit and adopt, not merely a name more consistent with our developing scope, but also a name which claims psychiatry as an art, as a practical and factual pursuit, not as a body of so-called universal truths, not (that is to say) as a science.

Upon this matter of *psychiatry as art, not science,* I should like to dwell. We must rejoice, along with our sighs of relief, in this of all years most contrasted with their predecessors, in this Victory Year, that the art of Marshal Foch came to outwit the pure science of the dead brains of old Clausewitz. Let us always remember medicine in general, and psychiatry in particular, as (in the words of Weir Mitchell 25 years ago to this very society) *an art with assistive sciences.*

To be sure, someone will each day be found to claim, as Clausewitz for war, so for medicine that it has at last become a science. But there is to the world a quality expressed by the late Benjamin P. Blood (William James's remarkable find here in the Americas) in the formula *Eternal Not Quite.* The world for Benjamin Blood lay (as you might say, gasping but exhilarant) in this principle of the Eternal Not Quite. Medicine may become almost a science, but never quite. Psychiatry may seem to harden into formulæ, but not ever quite. The cerebral cortex may seem to become a temple of mere sex or of just food; but the camouflage of sex in these remote neurones seems after all not absolute camouflage, and self-preservation seems scarcely to indicate all these beautiful dendrites and other paraphernalia of the accumulating nerve cells. So too Clausewitz, eagerly studying the pragmatic Napoleon, thought he had beaten the universe's *Not Quite* and found war a science to be applied to France *inter alia,* like a rubber-stamp. Napoleon himself had said he knew no more of war when he was through than when he began. It may be he *knew* no more, but war is not any more a matter of mere knowledge than anything else can be safely wrapped in formulæ.

The proper topic of this address has been something of a thorn in my flesh during the past year. Since all of us to a man have been distraught throughout this year, as possibly in no previous years of our experience, no doubt my fragmentary comments on the situation will be condoned. We have passed through three quarter-centuries that seem to us beyond doubt the most important

75 years in civilization. Not that there have not been still more important single and critical years than possibly any of the past 75, but taking all in all, the scientific and social products of these years have been the supremest ever seen. The concerns of this association touch the broad fields of medicine, of psychology, of sociology and of engineering. It can be no duty, as it is surely no power of mine, to discuss the history of these years after the manner of a Henry Adams. Borrowing from my microscopic training, I have, however, tried to reconstruct for myself, in the hopes of transmitting a little of the atmosphere to you, something of the historical situation at the turn of each quarter century, something perhaps of the topics of conversation amongst Americans of our sort in the years 1844, 1869, and 1894. It should be one's duty to derive from these cross-sections some inklings of what the future may contain. In the realm of prophecy, however, it were perhaps safer to rely on the fancy rather than on the reason and to think of wishes rather than hypotheses.

In pursuing this method of the historical cross-section of a given year one comes strangely upon traces of the illusion of the *déja vu*. Somehow one gets the impression that what happens one year might as well have happened 25 years earlier. The traffics and discoveries seem curiously all of a piece with respect to their originality. The years fuse into one, as microscopic cross-sections of an organ or tissue collapse in the mind into a colorless, flat, two-dimensional image, which has its pragmatic or cash-value in discussion and thought but has lost much of its reality.

There is even something of this effect of the taking out of the time-quality, with nothing left but a certain mystical quality of the events themselves, when one considers what happened on such a year upon just this day, June 18. For instance, June 18, 1815, occurred the Battle of Waterloo, and upon June 18, 1756, was the Black Hole of Calcutta. We stand thus 104 years beyond Napoleon's end, and we stand 163 years beyond a certain ethical question that faced some Englishmen. And if we chose to date back from to-morrow, we should find 704 years ago upon June 19, 1215, that Magna Charta was signed. Mr. Charles S. Peirce, perhaps the most original of American philosophers, from whom both James and Royce drew significant parts of their inspiration,

found that one of three sorts of plan on which the universe might be founded was the plan of absolute chance, that he called Tychism. I suppose it is almost absolute chance that Waterloo, Blenheim (June 18, 1800), the death of a great neuroanatomist, Vicq d'Azyr (June 20, 1794), and the Black Hole came on anniversaries of these particular days that we now meet. Or is it on the contrary an example of what Peirce termed Anangkism, a system of absolute necessity? If not, according to Peirce, the third possibility in ground plan of the universe would be what he terms Agapism. All three of these doctrines, Tychism, Anangkism, and Agapism are expounded in Peirce's celebrated paper on *Evolutionary Love*. Peirce, than whom there was no more profound logician, mathematician and thinker living in our American scene during the life of this association, felt on the whole inclined to subscribe to the third of these hypotheses, though he spent much labor on developing the doctrine of the world as a product of absolute chance. Somehow Peirce has always seemed to me the most American of all philosophers. He was, as you might say, with his Tychism, just the sort of thoroughgoing sport that the Yankee prides himself on being, whereas, with his agapistic doctrines, he worked into the world just that degree of *bonhomie* that the Yankee never quite can succeed in concealing. If it were possible to combine most intimately some of the elements of the chance doctrine of the universe, namely Tychism, with some of the elements of the love doctrine, namely Agapism, then we should perhaps find that Charles S. Peirce stood for being not merely a sport, but a good sport, and this is perhaps a sufficient description of the American end and aim.

If I say to you that 100 years ago the first steam vessel crossed the Atlantic and reached Liverpool June 20, on the anniversary of the third day of our association meeting, if I recall that the first telegraph line was set up from Washington to Baltimore in 1844, namely, in the year of the foundation of our society, if I recall that in 1869 the Suez Canal was opened and the first American trans-continental railway put through to the Pacific, and if I recall that 1894 was the year of Coxey's Army, you will get, no doubt, a strange and rather mystical impression that somehow all these are peculiar characters of the nineteenth century that forthwith fuse into one.

It is only when one goes back, say 500 years, that the metamorphosis seems terribly distinct, especially as regards America. Five hundred years ago (I spare you any deeper excursions into the bowels of time) we find in 1419 that the Norsemen in Greenland had been enslaved by the natives. Four hundred years ago Cortez began the conquest of Mexico and the mystical delight of our youth, King Montezuma, was killed in the same year (1519) that Magellan sailed. Betwixt the Greenland Norse slaves of 1419 and the Aztec conquests of 1519 there was certainly a huge apex of novelty projected upon the world. Three hundred years ago one coming from the Commonwealth of Massachusetts must remember that the Pilgrims were still in Holland. If one's germ-cells had another geography, one might think of the first families of Virginia as being already for some years laid down in their particolored way after the Jamestown settlement. Two hundred years ago, I find it of note personally that the colonists in Massachusetts were now just beginning to use tea and that in the selfsame year, 1719, *Mother Goose's Tales* were published in Boston. One could read those tales and drink the new tea only by hour-glass, as clocks arrived only next year (1720). Meantime as we sit here in Philadelphia, let us remember that William Penn had just died the year before, namely, in 1718.

One hundred years ago, not only was the steamboat only three days out of Liverpool, as I said above, and not only did the first steamboat make a trip on Lake Erie, but I find a number of other points in the annals of a very modern ring. The foundation of the Capitol at Washington was laid. The first national financial crisis developed following excesses in speculation. The hatters formed a union. Shoe pegs had just been introduced. Cyrus Field was born, but the Atlantic cable was still unsuspected. Elias Howe was born to construct the sewing machine. W. T. G. Morton was born for his tasks in ether anesthesia. There was veritably about to be a most pragmatic age if we are to agree with pragmatists that things consist in their consequences. Nor could the world afford to be unconscious of the birth in that year of Julia Ward Howe, of James Russell Lowell, and last but not least of Walt Whitman, most pragmatic, most democratic, most American of all. All of these were, by a simple mathematical calculation, 25 years of age in the year in which our own association was born.

Let us turn to that year, 1844. In that year the Royal Medical and Chirurgical Society was founded in England, though it had been chartered in 1834. The Sydenham Society had been founded in 1843, and the Pathological Society of London was shortly to be founded in 1846. Sir Charles Bell had died in 1842, though his great discoveries with respect to the differentiation of sensory and motor nerves dated many years back. Science, as a whole, was active. Faraday was, for the moment, resting between the period of his electrical discoveries and the period of his magnetic discoveries. It was in this very year that Charles Darwin wrote to Sir Joseph Hooker his celebrated letter—how he was now "almost convinced that species are not (it is like confessing a murder) immutable." In that year, too, Darwin laid away in his desk a 250-page manuscript which he was only compelled to bring out publicly in 1859 (at a time when Wallace's researches were maturing), namely, the basic material of the *Origin of Species*. Pasteur, meantime, was about his fundamental chemical discoveries, whilst Koch was in the cradle. These were the days in which Johannes Müller's researches in physiology were at the apex—days of Schwann, of Henle, of Purkinje, of Kölliker, as well as of Magendie, of Flourens, of Claude Bernard (who had found cane sugar in the urine, 1843) and of Broca, whose aphasia studies came to a head only, however, in 1861. Possibly someone might have mentioned the great thesis of Oliver Wendell Holmes, in 1843, on the contagiousness of puerperal fever, about which so rancorous a controversy was to rage for many years. Perhaps the researches of Braid on Animal Magnetism and his work on hypnosis which came out in the year 1843, called *Neurypnology or the Rationale of Nervous Sleep,* might have come to the attention of our Original Thirteen. Esdaile was not to have begun his surgical operations upon convicts with no other anæsthetic than hypnotism until the next year after our initial meeting, namely, the year 1845.

Many of the great men of our specialty in its early phase were now dead or their work completed. Pinel died in 1826, in which year Calmeil had given a description of General Paresis. Pritchard's work on moral insanity came out in 1835. Esquirol had died in 1838. So much of our modern psychiatry depends upon our knowledge of the venereal diseases that it may be of note to

recall that it was in only 1838 that Ricord had succeeded in subdividing the venereal diseases and dissolving the errors of John Hunter.

Our first quarter-century was that of Griesinger (1817-1868), perhaps the greatest of the systematists in psychiatry. In 1840 and 1841, Griesinger collected at Winnenthal the materials for the first edition of his book on mental diseases, which he was occupied with writing in 1844 whilst in private practice, and which was published in 1845 (several editions, to 1861) when Griesinger was 28. A year before the end of our first quarter-century, Griesinger died of a perforating appendix abscess at the age of 51 (1868). He had been planning a trip to America.

Let us prepare to leap forward a quarter-century from 1844. We leave the age of Faraday and the age of Wordsworth and Carlyle. In America there were Irving, Fenimore Cooper, and then Hawthorne. James K. Polk was elected president in 1844. Victoria had been ten years on her throne. Lafayette had died ten years before, Goethe, a dozen years before. O'Connell's trial might have been talked of by the Original Thirteen. Daguerreotype plates could have been made of the Thirteen, as the idea had been introduced in 1839. Bernadotte was King of Sweden, Louis-Philippe had not yet abdicated the throne of France (1848) and in this year visited England. In America the Puseyites were greatly in question; Millerites told of Christ's coming and the end of the world in October. The Mexican war was shortly to begin. Lieut. Ulysses S. Grant, now 22 years of age, was at his post in Louisiana. Texas was admitted to the Union as the 28th state. This year was the first in which Congress passed a bill over a president's veto. The national debt amounted to about sixteen millions. There were less than 79,000 immigrants and aliens in the United States. Morse offered his telegraph to the government at a low price, but the invention was deemed of little value. *Littell's Living Age* appeared in Boston and Robert Bonner established the *Philadelphia Ledger*. The *American Farmer* was issued in Baltimore. The *Essays* of Emerson were appearing, and one might be reading Cooper's *Afloat and Ashore* or the *Deer Slayer,* or on the other hand content himself with Longfellow's *Ballads*. That amazing contribution to civilization called Mormonism was on its way, and its founder Joseph Smith was shot that year in Illinois.

It is curious to inquire what the situation was with the movement that now interests us most. Marx's *Rheinische Zeitung* was suppressed in 1843, and *Vorwärts* was suppressed at Paris in 1845. The Communist manifesto was to come in the revolutionary year, 1848. Note well that these socialistic movements had begun prior to the great publications on evolution. Thomas Buckle, the last of the great historians before the doctrine of evolution, was 23 years old in 1844, and, although already one of the foremost chess-players of the world, had made up his mind to devote his life to history and had begun to spend ten hours a day in studies culminating in his *History of Civilization in England*, 1857-1861, a work many of whose conclusions sank into the every-day speech of popular thinkers and orators.

Herbert Spencer, meantime, was still an engineer on the London and Birmingham Railway, there acquiring the bent toward mechanics which runs through all his philosophical works in analogue after analogue. *Social Statics* was not to appear until 1850.

I have spoken of 1869 as the Suez Canal year and the year of the Union Pacific. These engineering feats were perhaps not the most important features of the situation. If you will look in the *Almanach de Gotha* you will find pictures of Ulysses S. Grant, who took presidential office in that year, and of Bismarck. The centenary of the great Napoleon was being celebrated in France, where of course Plon-Plon was emperor. The 21st Œcumenical Council was summoned that year, being 306 years after the 20th Council of Trent (1545-1563) to condemn Luther, Zwingli and Calvin. The patriarch of the Greek Church refused to attend the council. Pope Pius IX, who had been elected to office in 1846, celebrated a jubilee April 11, 1869. Temporal power was to be lost in 1870.

Faraday was now dead (1867). It was the adolescent period for the evolutionary theory, yet the *Descent of Man* was not to be published till 1871, and Spencer's principles of sociology were not to begin to appear till 1876. Crookes was engaged in investigations of psychic force and was to report in 1871. W. T. G. Morton, the discoverer of the uses of sulphuric ether, had just died, 1868, at the age of 49. In one of the year books I find a note on dynamite, which was invented in 1868, running as follows: "What

influence the new explosives, picrates, dynamite and ammonia powder will have on warlike operations remains to be seen." Anyhow, a torpedo school was set up by the United States at Newport.

Jenner's system of taking lymph from the inoculated animal was returned to, yet Pasteur's inoculations for hydrophobia were not to be undertaken until 1885.

That year Purkinje died at 82, Lamartine at 79, Saint-Beuve at 65, to say nothing of Jomini, the great formulator of the processes of war, at the age of 90. Livingstone was now lost in Africa, to be found in 1871. Grisi, the dancer, died.

I find in one of the books of scientific annals the following items, with reference to America from the scientific point of view. These annals record with astonishment the new American idea of hospitals for drunkards, of which it is said there were now four, Boston having led off in 1857, New York and Media following, and Chicago founding the fourth such hospital in 1868. The French account states that the victims come either voluntarily or dead drunk, perhaps having said adieu to the bottle in one last bout or perhaps having been made drunk for ease of transportation. In three months as a rule they went out cured. Tapering off was false treatment. It is of note in this connection that a national temperance convention was held in 1869 at Chicago and that the National Prohibition Party was organized, thereby sowing the seeds of a particular brand of mental hygiene, which promises so many developments with us to-day.

The annals remark also upon the tent-hospitals and tent-barracks, which were an American idea derived from the excellent results of tent treatment in the American Civil War. The French reporter remarks that the Germans had quickly caught up the idea from American practice and applied it in their own war of 1866.

A third reference to America is to the telegraphic transmission of a patient's pulse from Boston to Salem, where the vibrations were demonstrated by Upham on a lecture screen.

I note also from the contemporary annals the interest attaching to the discovery of three anesthetics analogous to chloroform; progress in the use of bromides in epilepsy by Legrand du Saulle; the new work on chloral discovered by Liebreich; the proof that absinthe could cause convulsions (Magnan). Clerk-Maxwell had

improved the zoëtrope, and Donders had perfected an instrument for measuring the rapidity of thought. A military surgeon, one Armand, was now making extraordinary claims for the virtue of smoking opium in all manner of nervous diseases, to say nothing of bronchitis and laryngitis. I note, too, an interesting and early announcement of what we might now term the mental hygiene of crime. The reporter states that waves of crime are spread by neuropaths. He states that these waves are not necessarily initiated by neuropaths, though spread by them, and he remarks that the phenomenon is important to the *Santé Publique*.

Let us turn to America. It was in May, 1869, that Lee repaired to Washington to consult with Grant. Custer was fighting with the Cheyennes at Wichita. John Lothrop Motley was appointed to the Court of St. James. The Horace Mann School for the Deaf was opened in Boston. George Peabody was giving munificent sums. $7,200,000 had just been paid (1868) for Alaska.

There was a great national peace jubilee and musical festival held upon two of the very days of our own meeting this year (June 15 to 19) in Boston. The chorus, they record, consisted of 10,371 selected voices, and the instruments, amongst them anvils, numbered 1094. Meantime that year the case of Jefferson Davis was nol-prossed. It was the time of the Alabama Claims. The KuKlux Klan list was issued. Jim Fiske and the Erie Ring stood in the public eye. The public debt was now two billion six hundred million, approximately, and the population of the United States was now over 38,000,000.

To me, perhaps the most picturesque event of this year was the exploration by Major J. W. Powell, of the Grand Colorado Cañon. To think of Powell in the cañon with his ten men in their four boats, and at the same time to think of David Livingstone in the midst of Africa, is to think of the year 1869 as not merely a great engineering year, of canals, railways, and cables.

Roebling was surveying for the East River Bridge. A 400-foot span bridge was thrown across the Ohio River at Cincinnati that year. Yet the incandescent lamp was not to appear till the year 1879, nor was the first electric trolley line to be developed till the decade 1880-1890. Whether the world attains the greatest speed in theoretical and practical researches and investigations may be doubted. I might have recorded that in the year before our

association first met an American named Starr had described in a British paper specifications for a sort of incandescent lamp, but Starr died suddenly at that time. Authorities differ on the question whether human progress can be accelerated by taking thought. Why was the airplane not developed a decade earlier than it was? Someone replies: "Because of the backwardness of motors." But why were the motors backward—because of some other lack of progress, theoretical or practical.

But to return to the 1869 situation, and in particular to the social set-up, upon which set-up it will be seen that in the end our interests converge. I have noted the organization of the National Prohibition Party. There was also a national labor convention held in 1869 at Philadelphia, and a woman suffrage convention was held. Boston is noted as the place in which co-operation in the labor movement had begun, so far back even as 1844, and I noted in that year the foundation of the Hatters' Union. *Das Kapital* had been issued by Marx in 1867, and in 1869 the great Bakunin (if I may be allowed the expression great for the greatest of anarchists) founded the Social Democratic Alliance, which was affiliated that same year with Marx's International Workingmen's Association. It was not to be until toward the close of the second quarter-century under discussion, namely, 1892, that Bakunin was outvoted and expelled from the combination, leaving anarchism and socialism to be henceforth distinct.

Bakunin and his ilk ought to be the special concern of the members of our association, who are, as you might say, almost alone in the possession of knowledge, whereby to understand the ins and outs and uttermost depths of ultra-individualism. Individualism, to be sure, is not found alone in Bakunin and the anarchists, but is found amongst many other world leaders, including the capitalists themselves. Bakunin and Bolshevism both begin with the letter B, and the history of Bolshevism dates back at least to 1869. We are accustomed to think of Bolshevism as a product of a deterministic philosophy and of a materialistic philosophy. From one aspect the evolutionary theory strikes some observers as both deterministic and materialistic; yet the chances are that a study of the evolution of Karl Marx's ideas would show them granted at least as much in the metaphysics of Hegel as in the ideas of Darwin.

John Stuart Mill published his famous essay *On the Subjection of Women* in 1869, and had already in 1867, during the discussion of the Reform Bill, advocated woman suffrage. In America the idea had been seething in the forties, and Dr. Elizabeth Blackwell had published her *Vindication of the Rights of Women* so far back as 1849, whilst the whole idea goes back over 125 years to Mary Wollstonecraft (1792). But again, in the suffrage movement, as in the prohibition question and as in the socialist question, a period of 50 years seems to have been required to bring these perfectly clear issues (at least as clear then as now) to a thoroughgoing consideration by the entire people and all parties. Even Fenian agitations were reported greatly in evidence in 1869.

Much then was doing in those years, much more than could have been talked at round tables in our association. Still in that year appeared Aldrich's *Story of a Bad Boy,* Bret Harte's *Outcasts of Poker Flat* and Mark Twain's *Innocents Abroad.* Whittier published his *Ballads of New England.* John Bascom got out some *Principles of Psychology. Lorna Doone* was published by Blackmore, and Herbert Spencer's *Data of Psychology* appeared. Dr. Oliver Wendell Holmes was defending his discovery of the stereoscope from certain priority claims. An association of American editors was organized. Horsford's baking powder was floated. Apomorphine was discovered by Matthieson of London. There was altogether much doing which required paper and ink, and a method was introduced in the year 1869 by which wood could be ground up to form the raw material of paper. In fact, the first post card appeared in Europe in Austria-Hungary in 1869.

Let us make our second saltation to the year 1894. This chosen cross-section method forbids us to look very far forward or back of the critical years of the quarter-century terms. But if we think of the first quarter-century as that of Helmholtz and Darwin, and as exploiting all the ideas for which Faraday stood, perhaps we can think of the second quarter-century as somehow standing most gloriously associated with the name of Pasteur. I fancy that the physicists and the chemists, to say nothing of the mathematicians, might find some other name than Pasteur's and that the illumination of the record with the discoveries of Koch should be as bright as any. But, somehow, Pasteur stands preeminent in the quarter-century of which we speak. Pasteur himself died

in 1895. It was with singular insight that Lavoisier was chosen by the French revolutionists 100 years before, namely, in 1794, for the guillotine. The French revolutionists, like some modern revolutionists, said that the Republic has no use for savants. For Pasteur, better one life than all the glories of war. Yet, however good and beautiful a sentiment was this of Pasteur, the better achievement was his pragmatic achievement, first in chemistry and then in bacteriology.

But from our own neuropsychiatric standpoint, perhaps this quarter-century is more that of Charcot. Charcot died in 1893. Brown-Séquard died in 1894. As for Charcot, time has rung in his changes with a considerable increment to Charcot's fame. Like every great functionalist, so far as I know, Charcot had begun his life with the hardest sort of organic work, and with no small achievement of concrete results both inside and outside the confines of the nervous system taken structurally. In his later years, he ventured into the more difficult paths of functional study, and with various men and in various places, achieved the fame of a charlatan for his pains; but it is not too much to say that without Charcot we should not have had either the Marie or the Janet or the Babinski of to-day, nor, I believe, the Freud, who early came under the Charcot influence. Just now in the war time, the ideas of Charcot seem to have been still more abundantly proved. Babinski and Froment have revived some of the Charcot ideas of reflex paralysis, and Nonne, the accomplished neurologist of Hamburg, has gone pretty far (for a German in wartime) in saying that experiences with war hysteria chiefly justify Charcot's views rather than those of others. With all the twaddle that limps forward, confusing dynamic with psychic, and refusing to make plain distinctions in the functional field, with all this, Charcot would have been entirely unsympathetic.

Much testimony from the war shows that Charcot was right in many of his oft-disputed contentions. In any event, the Salpétrière-Nancy controversies over hypnotism and suggestion seem to me to have left the deepest marks on the hardest strata of all psychiatry, viz. the so-called functional diseases.

I can be brief about happenings in 1894. Mark how social complications heap! There was Coxey's Army. The Lexow committee was at work in New York. The Elmira Reformatory was

being investigated. Crispi was shot. Fast Day was abolished. Instead, Congress made Labor Day a legal holiday. The American Order of Steam Engineers met at Baltimore and stood for individual rights against strikes and boycotts. The Hudson Bay Company gave up its charter to the Canadian Government.

The Germans erected a monument to Blucher, at his Rhine-crossing; and the Emperor Wilhelm unveiled a monument at Königsberg to his grandfather. It was 25 years before (1869) that the first great military post was set up at Wilhelmshafen. The new Tower Bridge, meantime, was opened in London (June 30). Less than a month later (July 29) the Emperor Wilhelm arrived at Dover on the yacht *Hohenzollern*. He was of course cordially welcomed. In November Nicholas II was proclaimed Czar of Russia and of Poland, and Grand Duke of Finland. Japan had a war with China. President Carnot war assassinated in France June 24. The great Lyons exhibition of arts, sciences, and industries was opened. King Leopold of Belgium opened the World's Exhibition.

My task was to speak of an anniversary. I have adopted the device of cross-sectioning the years, no doubt at all too brief intervals in so long a history and beyond question choosing facts in quite too random a fashion. Yet the variety and the heterogeneity of the facts and the arbitrariness of the trisection allow, with all the greater certainty, a number of conclusions and comments. These I shall set forth with a baldness quite unjustifiable save by the brevity of our time.

1. The American Medico-Psychological Association, now over 900 members strong and representing a large majority of the United States and the Canadian provinces, being the oldest national medical association in continuous existence (so far as we are aware) on the continent, has a history of 75 years, cast in a time of almost unprecedented interest in the world's history to date.

2. During these 75 years an extraordinary process of public enlightenment concerning mental disease has gone forward, *pari passu* with general progress in education, and the more material and engineering sides of economics.

3. Put in a phrase, this progress has been to a deeper and more pragmatic hygiene in all matters pertaining to the mind. Perhaps the most eminent of our earlier members, Dr. Isaac Ray,

was the author of a work on *Mental Hygiene,* in which there was, from our present viewpoint, much elaboration of the obvious and in which there was naturally very little of the modern social conception. Yet Ray himself was one of the founders of the Social Science Association and distinguished himself, as Dr. Charles K. Mills this morning said, by writing an excellent work on the *Jurisprudence of Insanity.*

4. Just as Ray's *Mental Hygiene* was largely devoted to a consideration of individual psychiatry and took up the psychiatry of the person as such and as affected by various conditions of the society in which that person's life befell, so on the other hand Ray's *Jurisprudence of Insanity* dealt with what we would now call forensic psychiatry, that is, with public or governmental aspects of mental disease: accordingly the whole intermediate realm of social psychiatry proper, that is, of psychiatry that deals neither with the individual person as such nor with his legal or institutional relations, got no formulation in the early years of our association's life.

5. As Isaac Ray typifies our membership 1844-1869, so perhaps Edward Cowles typifies the membership in the second quarter-century of our association's existence. Cowles stood—and thank God still stands—for a profounder insight into the nature and causes of mental disease and defect, and no doubt to him is greatly due the impetus to the establishment of laboratories in our institutions. This is no place to eulogize the living. But the third quarter-century, now coming to a close, could not have been so greatly distinguished by the laboratories and by the exercise of what has been called the *laboratory habit of mind,* had it not been for Cowles. Nor is this a personal view of my own. A dozen of the best men amongst our psychiatrists have said as much to me in the last few years.

6. Perspective interferes overmuch with our estimate of a typical personality for the third quarter-century. I myself believe that no greater power to change our minds about the problems of psychiatry has been at work in the interior of the psychiatric profession in America than the personality of Adolf Meyer. If he will pardon me the phrase, I shall designate him as a ferment, an enzyme, a catalyzer. I don't know that we could abide two of him. But in our present status we must be glad there was one of him.

No American theorist in psychiatry of these and the immediately succeeding decades but is compelled either to agree or else—a thing of equal importance—most powerfully to disagree with him. And who shall say that anybody is abler to get truth and reality out of disagreement and error than psychiatrists?

7. The outstanding development in the latter years and especially in the last quarter-century of the association's history has been, to my mind, the development of social psychiatry, than which it might be hard to name a more important feature of the face of the world to-day. Social psychiatry, even were we to include (what practically is not included, namely) public psychiatry within its conception, is far from the whole of mental hygiene. For mental hygiene includes also the far more difficult and intriguing topic of the psychiatry of the individual, as related to himself and his organs and processes.

8. Personally I hold, and I think every physician and especially every psychiatrist must hold, that the individual is not only the unit of the physician's interest, but also (following Herbert Spencer) the unit of the sociologist's interest. This we ought to maintain, I think, against the supposed sociological improvement introduced by Schäffle, namely, that the family is the social unit. Accordingly I hold that the foundation of social psychiatry (as also of public psychiatry) is the psychiatry of the individual.

9. Now it was just at the outset of our third quarter-century that Josiah Royce made his theoretical contributions to the conception of the social consciousness (1894-1895). From that atmosphere developed in the work of Richard Cabot the idea of medical social work. Mark you that this idea was far more than a mere addition of two ideas, namely the idea *medicine* and the idea *social work,* but was a productive combination of these ideas, an actual novelty. It was then only a step to the development of psychiatric social work in Massachusetts, 1912, a step stated by Cabot himself (at the recent meeting of the National Conference for Social Work) to be the greatest innovation in medical social work since its foundation.

10. From Bakunin to Lenin is a half-century. What has the world to say of anarchism and Bolshevism? Certainly these are no new things. Perhaps neither Bakunin nor Lenin is a topic for alienists of the old medicolegal group: These world

leaders are not on the minute to be interned as insane! But does any man of us here believe that the psychiatric view-point could fail to throw light on Bakunin and on Lenin? Alone amongst the specialties of medicine, psychiatry has for its daily task the consideration of the entire individual. The rest of the branches of medicine, even neurology, appear to remain much too analytic in their view of a man. Psychiatry alone uses the daily logical apparatus of the synthesizer.

11. Is Mental Hygiene ready for the problem of Bakunin and Lenin? Alas! No! We have our "Varieties of Religious Experience," but no James has arisen to depict, on the basis of the extremest cases, the varieties of political experience. In fact the delineator of Lincoln or of Roosevelt as in any sense psychopathic might well bring down upon his head far more partisan fury than one who should discover the queerest traits and episodes in religious heroes. We deal with *Aqua Regia,* with Damascus blades, in our psychiatric laboratories and armories. "Divide to conquer" is a necessary precaution. We must teach the world, what we as physicians have so recently learned, namely, that to be crazy is to be one of scores of things. To describe Lincoln as a cyclothymic with attacks of depression or Roosevelt as constitutionally hyperkinetic (always supposing these to be true designations) should be no more impolite or less objective than to think of Bakunin or Lenin as paranoic personalities. Crazy? No! But, cyclothymic or paranoic, certainly!

12. Insanity is mental disease, but not all of it or rather of them. Alienists are psychiatrists, but not all or in the long run the majority of psychiatrists. "Alienistics," as we may call the doctrine of medicolegal insanity, is not the whole of psychiatry. But, above all, psychiatry must be conceived to include the minor psychoses, the smallest diseases and the minutest defects of the mind as well as the frank psychoses and the obvious feeble-mindednesses. The psychiatry of temperament is an art that might fling itself very far. Mr. Wilson, I believe, spoke of some members of his cabinet as temperamental. As a cat may look at a king (time and weather permitting), so I suppose a psychiatrist might look at a cabinet officer, at least in one of his temperamental phases.

13. We passed from the age of Darwin to the age of Pasteur to the age of Metchnikoff and of Ehrlich. We lived through the

beginnings of systematic psychiatry in the period of Griesinger, we witnessed the first clarifications of mental disease function in the period of Charcot, and we have just concluded a war whose psychiatric achievements (from the deepest theoretical side) trace back to Charcot, flowering to my own mind in Babinski. In America, outside institutions, there had been a dearth of great theorists after Benjamin Rush. But the basic ideas of Weir Mitchell were no doubt being laid down in the war time of our first quarter-century only to effloresce in the second period. The work of Charles K. Mills stands out for me as of the greatest theoretical importance in American work in that second period. I think of Donaldson as a great force in our third period, if we are looking outside institutional ranks.

14. But it is clear that the American idea Mental Hygiene must have grown in philosophic circles too. I think first of the great Emersonian period, with its grotesque parody called Eddyism or Christian Science. Then I think of the laying-down of the idea of pragmatism by Charles Peirce, the great and little known central figure of American thought. And then I think of the man William James who put pragmatism across the American scene, but added thereto what I may call *the psychiatric touch* and really typifies all that is best in American thought. Emerson, Peirce, James—these are three American names to conjure by, and they are deeply responsible for the spiritual, the logical, and the practical factors in the whole of mental hygiene. With their spirit, illumination and dynamism, we shall face the terrible analyses of the present hour—the rights and interests of the individual as against society and of society as against the individual—with full confidence that synthesis will follow analysis and the task of Humpty-Dumpty solved at last.

15. Do you not agree with me that in all the *pot-pourri* of the years this great problem of the place of the individual stands out? That American thought, transilluminated as always by the softened European lights, contains within itself immortal fundaments of the mental hygiene of nation, race, and person? And may we not rejoice, as psychiatrists, that we, if any, are to be equipped by education, training, and experience better than perhaps any other men to see through the apparent terrors of anarchism, of violence, of destructiveness, of paranoia—whether these tendencies are

shown in capitalists or in labor-leaders, in universities or in tenements, in Congress or under deserted culverts? It is in one sense all a matter of the One and the Many. Psychiatrists must carry their analytic powers, their ingrained optimism, and their tried strength of purpose into not merely the narrow circles of frank disease, but, like Séguin of old into education, like William James into the sphere of morals, like Isaac Ray into jurisprudence, and above all into economics and industry. I salute the coming years as high years for psychiatrists!

LXI

APPLICATIONS OF THE PRAGMATIC METHOD TO PSYCHIATRY

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APPLICATIONS OF THE PRAGMATIC METHOD TO PSYCHIATRY*

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WHAT difference would it practically make to anyone if this notion rather than that notion were true? If no practical difference whatever can be traced, then the alternatives mean practically the same thing, and all dispute is idle": such is one of the briefest formulations that Professor William James ever made of the pragmatic method. Again, there can be "no difference in abstract truth that doesn't express itself in a difference in concrete fact, and in conduct consequent upon the fact, imposed on somebody."

That the pragmatic method, altogether aside from any philosophic implications, must have very important relations to medicine would nowadays be denied by no man. In fact, the whole attitude of the medical man to medicine would not suffer if it should receive the designation "pragmatic." The very founder of the idea of pragmatism, Charles Peirce, used to speak of it as the "laboratory habit of mind." Pragmatism in medicine must have especially close relations with treatment and prognosis. This can be clearly seen from one of the better and longer statements of the nature of pragmatism as given by James in Baldwin's *Dictionary of Philosophy and Psychology*: Pragmatism is "the doctrine that the whole meaning of a conception expresses itself in practical consequences, consequences either in the shape of conduct to be recommended or in that of experiences to be expected, if the conception be true; which consequences would be different if it were untrue, and must be different from the consequences by which the meaning of other conceptions is in turn expressed. If a second conception should not appear to have other consequences, then it must really be only the first conception under a different name. In methodology it is certain that to trace and compare their respective consequences is an admirable way of establishing the differing meanings of different conceptions."

*Read in abstract at the Thirty-fourth Annual Meeting of the Association of American Physicians, Eleventh Triennial Session of the Congress of American Physicians and Surgeons, Atlantic City, June, 1919.

It is my object in the present paper to apply pragmatic principles to some problems of psychiatry.

1. PSYCHIATRY AND CLINICAL NEUROLOGY

In the first place, does psychiatry exist as distinct from neurology? It can be pointed out that the research basis of psychiatry and of clinical neurology is a unit, namely, theoretical neuropathology, considered in both structural and functional aspects. Let us, however, apply the pragmatic method. Is psychiatry only clinical neurology under a different name? The moment we trace and compare the respective consequences of these two ideas, we perceive that clinical neurology leads to a quite different effect upon the patient from that to be expected when psychiatry approaches him. Psychiatrists and clinical neurologists have had no identical education as clinicians, and they have not the same outlook. This is a practical situation which has pragmatic consequences. It is simply a pious wish when we state that clinical neurologists and psychiatrists are all really nothing but neuropsychiatrists. Perhaps they ought to be and perhaps they soon will be, but they are not. It makes a great deal of difference to the patient whether he is approached by a psychiatrist or by a clinical neurologist.

I have elsewhere gone into more detail upon this matter, referring especially to the different points of view, e.g., toward mild cases of schizophrenia (dementia precox) and cyclothymia (manic-depressive psychoses), maintained by neurologist and psychiatrist respectively. I will not here discuss the point further.

Clinical neurology and psychiatry are pragmatic entities, for they have different consequences when their points of view are applied to the victims of disease.

2. THE SO-CALLED "UNITY OF INSANITY"

Turning to the subject matter of psychiatry, the largest question hangs upon whether we define mental diseases as a unit or as a set of entities. English writers, among them some of the more subtle, such as Charles Mercier, have for years plumped for the unitarian view. One must have a certain respect for any Anglo-Saxon view of the topic, seeing that it no doubt has a certain relevance and practical applicability. However, I think the progress of psychiatry proves, not only that mental diseases can be analytically split up into logical entities, but that these logical units have pragmatic value. At the same time that a thinker like Mercier insists upon the "unity of insanity," he also insists upon grades of responsibility in medicolegal cases and claims to be one of the earliest to have made this distinction. In fine, Mercier lugs in at the finish what we practically want in the matter of subdivisions within psychiatry. If we look more narrowly at other British authors, we find that they often dispose of the unity question in mental diseases by throwing into the field of "nervous" diseases everything which does not handily fit a preconceived legal definition of insanity. This again may comport with Anglo-Saxon notions of practicality and may have a certain relevance from the standpoint of law courts. But assuredly we do not get on very measurably in psychiatric research, if we simply redistribute our material to suit some nonmedical criterion, such as some standard of the practical responsibility of mental cases.

It seems to me that the progress of psychopathic hospitals, in the few places in the world where they have been rigorously tried out, shows beyond cavil that psychiatry is an art dealing with numerous entities. Possibly (according to a rough enumeration from popular systematic works) there may be between eighty and one hundred of these practical entities.

The pragmatic question is, what difference does it make to the patient whether he is said to be affiliated with one or the other of these entities? I am not sure that I can prove that there is a different "conduct to be recommended" or different "experience to be expected" (to use James' phrases) for all of these eighty-odd entities. But I am entirely sure that it makes a great difference to the patient whether he is to be placed in one of ten or a dozen major groups of these entities. Perhaps I entirely miss the point of the British contentions for the unity of insanity; but I really can not see that their viewpoint has a leg to stand upon when we contemplate the present nosological situation. It certainly makes a great deal of difference to the patient, as well as to the social unit in which he lives, whether we regard the patient as a victim of neurosyphilis or whether we regard him as a victim of alcoholic psychosis. Now, possibly the English authors would wish to exclude the victim of neurosyphilis and of alcoholic mental disease from the realm of insanity altogether. Very well,—let this happen. We should then at best attain a conception of the unity of insanity only by greatly diminishing its contents, as ordinarily conceived. But would any author deny that it makes a great deal of difference to the patient whether he is a victim of psychoneurosis or of some depression falling in the group termed by Kraepelin manic-depressive psychosis (which I think might as a group better be termed the cyclothymic group)? Now again, if the unitarian wishes to diminish the field of insanity by cutting off the psychoneuroses, so be it. Again we shall attain unity by a Procrustean process. The psychiatrist will give up any insight he may have into the psychoneurotic and hand his patient forthwith over to the clinical neurologist. Yet the research basis for the understanding of psychoneuroses is no doubt identical for both the psychiatrist and the clinical neurologist. Again the pragmatic method should be applied. If it is true that it is going to make a great difference to the patient whether he is regarded psychiatrically or neurologically, then we must decide that there are real differences under discussion.

Parceling out the psychoneuroses and the cyclothymias to the neurologists and the psychiatrists, respectively, may seem logically the right thing to do. Pragmatically I believe it will have, not only practical, but evil consequences if we execute this partition.

3. DIAGNOSIS BY ORDERLY EXCLUSION

Dismissing the question of the unity of insanity, I wish to approach a few other pragmatic questions in psychiatry and to hang them upon the order concept. Last year I advocated before the Association in a paper *"Diagnosis per Exclusionem in Ordine*: General and Psychiatric Remarks" the application of the principle of order in psychiatric diagnosis, and called attention to the existence of eleven major groups of entities which seemed to me to have practical value. Of course I was aware that the principle could be applied to many other branches of

medicine, and I am glad to say that in correspondence with Dr. Richard C. Cabot, we have together concluded that the principle might very effectively be installed at an early date in many of the specialties, if not in the whole of medicine. As Dr. Cabot has said to me, if one gets hold of a *list* of things it is manifest that said list may be turned into a *sequence* or order and that this sequence or order may have practical value. I shall not here repeat the considerations of the above-mentioned paper, at least to any extent; but I wish to claim that the application of the order concept is an example of pragmatic method. It makes a difference to the patient both in "conduct to be recommended" and "experience to be expected" whether we approach him diagnostically in an orderly fashion.

4. NEUROSYPHILIS AND GENERAL PARESIS

It has in the past made a good deal of practical difference to many psychopathic patients that they have not been primarily considered as possibly victims of neurosyphilis. Time was when the Wassermann serum reaction was taken only in cases in which one suspected general paresis. Nowadays so protean have been proved to be the forms of neurosyphilis that for practical purposes no mental patient can fail to be thought possibly syphilitic. That hypothesis must be disposed of one way or the other.

But if neurosyphilitic, what form of neurosyphilis is in play? It makes a great deal of difference to the patient whether we set up the hypothesis that he has general paresis (with all the evil prognostic connotations of that entity) or whether we limit our initial idea to his being merely neurosyphilitic. An application of the pragmatic method to the orderly diagnosis of the subentities of the neurosyphilitic group would make us consider at some point the *nonparetic* forms of neurosyphilis. We should, in the pragmatic interest of the patient, keep our diagnostic ideas in solution at the level of undifferentiated neurosyphilis rather than leap to the diagnosis "general paresis." We have practically seen a great deal of evil happen to patients by reason of these premature leaps to the diagnosis "general paresis," on the basis of a few clinical similarities to the entity as described in books. There is, therefore, much scope for the application of pragmatic method in the field of neurosyphilis.

5. DIAGNOSTIC PRECESSION OF "FOCAL" VERSUS "SYMPTOMATIC" CASES

To facilitate the rest of my discussion I will here reproduce the eleven groups of mental disease from my paper on "*Diagnosis per Exclusionem in Ordine.*"

Mental Disease Groups (Orders)

I. Syphilitic	Syphilopsychoses.
II. Feeble-minded	Hypophrenoses.
III. Epileptic	Epileptoses.
IV. Alcoholic, drug, poison	Pharmacopsychoses.
V. Focal brain ("organic," arteriosclerotic)	Encephalopsychoses.
VI. Bodily disease ("symptomatic")	Somatopsychoses.
VII. Senescent, senile	Geriopsychoses.
VIII. Dementia precox, paraphrenic	Schizophrenoses.
IX. Manic-depressive, cyclothymic	Cyclothymoses.
X. Hysteric, psychasthenic, neurasthenic	Psychoneuroses.
XI. Psychopathic, paranoiac, et al	Psychopathoses.

Why is Group V of the Encephalopsychoses placed prior to Group VI, the Somatopsychoses? Logically, it might well seem that we should approach, in the process of diagnosis, the somatic diseases that give rise to mental symptoms prior to the focal brain diseases that produce mental symptoms. But logic is not practice. In the general hospital clinics, where somatic diseases, nonnervous in nature, are habitually approached logically prior to the nervous and mental diseases, we have not found much progress in psychiatry or much grasp of the psychiatric point of view. Mental cases will for many years to come in our hospital practice, devolve more seriously upon the attention of neurologists and psychiatrists than they will upon the attention of internists. If a man has mental disease and if one has excluded the great groups of syphilis, feeblemindedness, epilepsy, and alcoholism (with all their practical social implications), then one naturally desires to clear up other large brain aspects of this mental case. Although we are in possession of numerous data to show that mental symptoms may be merely symptomatic of bodily (nonneural) diseases, yet there is no doubt that the nervous system exerts in some ways a prior claim on the attention. Perhaps the practical answer would be that in internists' clinics, the order of logical consideration should be reversed and Group VI placed before Group V, whereas in psychiatric clinics the order might remain as stated. Let me insist at this point on something which is logically clear, but practically not always envisaged. *This system of orderly exclusion in diagnosis has nothing whatever to do with the practical order in which data are collected.* It is a plan to be put into effect *after the data are all in hand.* It does not matter whether the data about the pupils or the reflexes are chronologically collected before the data concerning bodily infection. The question is one of logical, not of chronological, priority. In the end, however, I am willing to acknowledge that the question, whether the encephalic or the nonneural somatic conditions should be diagnostically placed in the order I recommend, is an open question. The question can be answered in the long run only pragmatically.

6. DIAGNOSTIC PRECESSION OF SCHIZOPHRENIC VERSUS CYCLOTHYMIC CASES

One other example may be offered looking in the same direction. I have placed the schizophrenoses (dementia precox, etc.,) in Group VIII, ahead of the cyclothymoses (manic-depressive, etc.), Group IX. Now it might be pointed out that as manic depressive phenomena are more nearly normal than schizophrenic phenomena, it should be well to consider the patient normal before we consider him abnormal. Let the patient be regarded as innocent of mental symptoms before he is regarded as guilty of such. Well, the pragmatic answer to this question is that it appears to have important practical consequences whether we consider a patient schizophrenic logically prior to our considering him cyclothymic. A group of schizophrenic symptoms is much more convincing as to a patient's being a victim of the disease schizophrenia than is a group of cyclothymic symptoms convincing as to his being a victim of cyclothymic disease. Just in virtue of the quasi-normality of the ups and downs in mood of the manic-depressive does it transpire that practically every form of mental disease is capable of showing these ups and downs in mood. By consequence

if we fix our minds upon cyclothymic phenomena, we are rather inclined to premature fixation of our minds upon the diagnosis of the entity manic-depressive psychosis. Seeing cyclothymoid symptoms, we rush to the idea of the cyclothymic entity. In short a group of schizophrenic symptoms is much more "pathognomonic" than a group of cyclothymic symptoms. It was upon this pragmatic basis that Group VIII was placed before Group IX.

Why should we place the psychoneuroses in a Group X, following the schizophrenias and cyclothymias? I am bound to admit that this placement was an exceedingly practical one. We have in psychopathic hospital practice seen so many cases in which the diagnosis "nervous prostration" has been affixed to a patient who was really a mild example of schizophrenia or of cyclothymia (and this much to the therapeutic detriment of the cases in question) that it seemed wise to relegate the psychoneuroses to a subsequent position in the diagnostic list for orderly exclusion.

7. PLACEMENT OF INVOLUTION-MELANCHOLIA

Another example of the application of the pragmatic method is in the question of involution-melancholia. Controversy rages as to whether the involution-melancholias belong in the senile group (in my own arrangement, Group VII, the Geriopsychoses) or whether they belong in the cyclothymic (manic-depressive group) Group IX of the above list. Logically there might be some argument for placing the involution-melancholias among the senile and presenile conditions. The very *term* involution suggests this. However, it seems to me that we must grant that we know next to nothing (and pragmatically I should say absolutely nothing) about the etiology and genesis of involution melancholia. No doubt there is a darkling idea that involution melancholia is somehow an endocrine affair. We know nothing about the etiology and genesis of manic-depressive psychoses. But here the pragmatic method allows us to employ even our ignorance to advantage. There is not the slightest doubt that to place a case in the senile or presenile sub-groups of the Geriopsychoses is to give the case an evil prognosis with respect to tissue destruction and degeneration, perhaps unjustly and harmfully.

To place the involution-melancholias in the senile-senescent group is to cause certain experiences to be expected. Now, practically, we know that such experiences are not always to be expected in involution-melancholia, in short that clinical deterioration is not always found to suggest brain tissue degeneration. Pragmatically speaking, therefore, it seems to me that the reply as to the placement of the involution-melancholias is unconditionally to place them in the cyclothymic group, as compared with the senile-senescent group — and this despite our dense ignorance of the nature of involution-melancholia.

SUMMARY

1. Psychiatry should more and more adopt the "Laboratory habit of mind," become more and more pragmatic, and bring itself in line with the rest of medicine.

2. Seven applications of the pragmatic method to psychiatry are offered:

(a) It makes a difference to the patient whether he is seen by a psychiatrist or by a clinical neurologist: There is thus for the moment a real difference between psychiatry and clinical neurology, though the future may destroy that difference and produce "neuropsychiatry."

(b) It makes a difference to the patient whether we take "insanity" as a unit or as a collection of entities: The pragmatic rule decides in favor of a pluralistic view of mental diseases.

(c) The principle of *orderly* exclusion in the diagnosis of complicated cases is of pragmatic value.

(d) Especially is this true of the diagnostic field of neurosyphilis, where it is important to maintain the *non*paretic hypothesis as long as possible in the interest of the patient's therapy.

(e) Opinions might differ as to the advisability of entertaining the hypothesis of focal brain disease before or after the hypothesis of somatic (non-neural) disease in a given case: The pragmatic rule might decide one way for general hospital clinics and the other way for mental clinics.

(f) Schizophrenia should be eliminated before cyclothymia on the pragmatic basis, for a group of schizophrenic symptoms is much more decisive for dementia precox than a group of cyclothymic symptoms is decisive for manic-depressive psychosis.

(g) The pragmatic method decides that in the face of complete ignorance of its true nature, involution-melancholia is better placed in the cyclothymic (manic-depressive) group than in the senile-senescent group, if it is to be placed in either group.

LXII

THE ENGINEERING FOUNDATION

THE MENTAL HYGIENE OF INDUSTRY

A MOVEMENT THAT PARTICULARLY CONCERNS EMPLOYMENT MANAGERS

By
E. E. SOUTHARD, M.D.

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The Mental Hygiene of Industry

A Movement That Particularly Concerns Employment Managers

By E. E. SOUTHARD, M.D.

In February, 1919, *Engineering Foundation undertook to support a limited study by* Dr. Southard *of mentally abnormal persons in industry. This paper is an introductory statement to the records of this study, which is still in progress and is producing results of practical value. The term mental hygiene is used to cover the expert activities of medical men who deal with the problems of mental diseases, of those who are endeavoring to improve the methods of mental testing and of those persons in industry who are dealing with personnel matters and have had experience in character handicap cases.*

Dr. E. E. Southard *has been pathologist to the Massachusetts Commission on Mental Diseases since* 1909 *and Director of the Boston Psychopathic Hospital since* 1912.

"MENTAL hygiene of industry" is proposed as the term for a rapidly growing group of ideas suggested by such phrases as: *the human element, personal factors, individualization, character analysis, scientific selection, social significance, moral values, workman's standpoint, workman's ambition, creative impulse, instinct of workmanship, role of habit, fatigue and efficiency, anti-social behavior, wasteful emotions, unemployment and personality,* the *psychopathic employee, civilian shell-shock analogues, neurasthenia, a disease in engineers.* All these expressions and many like them have been found in recent engineering literature, to which an increasing number of articles is being contributed both by practical managers who have grown up in the work, and by specialists from many fields. The term has become familiar on account of the excellent results obtained in various fields in war and peace by the National Committee for Mental Hygiene, founded

in 1909, and has the special advantage of not assigning the human element to any one particular science or art such as psychology, psychiatry[1] or social work. The psychologist, whether inside or outside of a laboratory, can and often does correctly claim, that he is a mental hygienist. The psychatrist, neurologist or other specialist in nervous and mental diseases[2] can also properly term himself, and often does, a mental hygienist, and the newly developed art of social work, particularly in its psychiatric division, is developing new workers who are effective aides. Also the practical and intelligent employment managers of the present day, whether trained by experience alone or in the special courses recently established (and, of course, greatly stimulated by the war emergencies), are workers that also deserve the name, mental hygienists.

Time was when the mental hygiene of industry was regarded as hopelessly vague, nay utopian. Of course, the field of industrial hygiene itself has come under cultivation only recently. The world has become accustomed to the idea of public health, and it was only natural that the great conceptions of public health should begin to be applied to industry. Factories and mines began to lay their health problems at the door of public health specialists and other medical men. Meantime eager laboratory workers were more and more minded to carry their results from the test tube, the microscope and the smoked drum back to the factories and mines. Yet there was always over and under and penetrating all through these problems of factory and laboratory this so-called human element, baffling but inviting, vague but insistent. Probably the war served to precipitate out of vagueness the mental hygiene of industry. In the war the psychologists got interesting results with their individual and group tests. At the same time the specialists in nervous and mental diseases were

[1] *Psychiatry*, the name ordinarily given to the practical branch of medicine that deals with the diagnosis and treatment of mental diseases. The ending, *iatry*, refers to the matter of treatment or "healing." See also notes on terms at end of paper. *"Psychosis"* refers literally to any condition of mind, or mental state, but is ordinarily used by practitioners of medicine to mean a form of mental disease. Thus a victim of dementia praecox (see footnote No. 5) or of general paresis (see footnote No. 7) or of any other mental disease is said to be suffering from a psychosis.

[2] See notes on terms at end of paper.

showing concrete results in obtaining an army relatively free from liability to what (for the want of a better name) has been called shell-shock. Not the least achievement in the mental hygiene of the war was the development of a small but effective group of social workers as skilled as laymen can become skilled in the approach to mild mental and nervous diseases and their practical handling under medical direction outside institutions. When we use the term mental hygiene of industry, we intend, therefore, very definitely to suggest that something practical in the shape of a new art has been found, looking toward betterment in industry.

One good way of finding out whether a science or an art is really something definite, and not merely an empty logical fiction is to note whether the supposed science or art has itself developed a personnel. Let me briefly sum up the situation as follows:

(a) There is a *psychology of industry* because there are certain applied psychologists, or "mental testers." This applied psychology uses mental tests and scales (after the manner of Binet and others). These tests and scales have been developed for individuals in groups and are susceptible of vast specialization as industrial problems get definitely in mind. The mental tests seem to be especially useful in hiring. Perhaps they will become useful in the always difficult problems of promotion. Perhaps even some of the problems of job analysis for individual workmen will be solved by applied psychology.

(b) There is a *psychiatry of industry* because there are certain psychiatrists and neurologists who have actually been at work on the problems of industry. Its immediate value is likely to be in the problems of discharge and in grievance problems, particularly if by the term "grievance" we could be understood to mean also all sorts of minor dissatisfactions in plant management. Industrial psychiatry is likely to be of especial value, it would seem, on some sides of the analysis of "turnover." Practical experience in getting jobs for the mentally handicapped unemployed seems to indicate that industrial psychiatry will be of special value in job selecting for certain psychopathic or cranky or "different" employees, whom it may be extremely desirable to retain in service. Experience in this field will eventually

throw much light on the problems of hiring and promoting.

(c) There is a *field of psychiatric social work in industry* because there are psychiatric social workers now prepared to enter the field of industry; (a few individual workers have already entered the field, but too few from whom to draw general conclusions). Psychiatric social work is a new art, in part auxiliary to medicine, in part of independent scope. These workers track down the discharge, grievance, dissatisfaction, and psychopathy problems outside the factory or mine. They might help to track down these same problems inside the plant, on demand of employment managers or minor executives. The point of view combines "efficiency" and "welfare" and brings to the surface many stabilizing and destabilizing factors inside and outside the plant, factors that the managers have rarely seen till too late or perhaps in too general terms.

Progress and Limitations

Concerning (a), the new *industrial psychology,* there is no doubt of its permanence. Early claims were too sweeping; but, though the professors were overenthusiastic, the industrialists were equally injudicious in their expectation of readymade results and rubber stamp devices for manipulating the human mind. A little perspective or ordinary business sense should have stopped such vagaries.

But the personnel work of the psychologists in the American Army and the elimination, by neuropsychiatrists supported by psychologists, of the feebleminded from the Army have settled for all time the question of the applicability of skilfully and specially devised mental tests to groups of men as well as to individual men. We do not need to grant one-half of the claims made for this work to concede that this kind of mental-measurement psychology has come to stay. Even if we limited consideration to the personnel work of the Secretary of War's office alone, or to the work of the nervous and mental division of the Surgeon-General's office alone, we should be able to demonstrate the value of these methods. Of course, it will be a long time before the full story of these efforts and results can be properly told by the experts engaged. But no one who aided in the unheroic but strenuous "Battle of Washington"

and saw on the ground the progress made possible by the above two agencies can fail to see that a long step was there taken in military psychology. Here was a large scale production with a vengeance. It takes but half an eye to see that many of the methods and some of the conclusions of military psychology can be carried over with due modifications into industry. And in point of fact some of the army psychologists are now entering the industrial field.

Concerning (b), *industrial psychiatry,* the situation is undeveloped but hopeful. There are important lessons from the war here also. The Surgeon-General's Office had a busy and effective new division—new to any army it is believed—called the division of neurology, psychiatry and psychology. This combination of interests already constituted a long step in the direction of a true mental hygiene. To be sure, the sociological side of the problem was not adequately represented in the new division of the Surgeon-General's Office as it was constituted in the war. But that was, no doubt, due to the lack of practical sociologists interested in the relation of man-to-man, soldier-to-soldier. However, many of the psychiatrists and psychologists were sufficiently men of the world or socially minded that a due admixture of the sociological point of view was almost attained. In the end, too, the establishment of the so-called "morale officers" shows very well the trend of the whole plan toward a consistent military mental hygiene, representing psychiatric, psychological and sociological viewpoints.

If there was a military psychiatry entirely aside from the problems of the front, *i.e.,* a military psychiatry due to the existence or development of more or less mild and incapacitating nervous and mental diseases in American camps already on this side of the Atlantic, it is plain that an industrial psychiatry must exist of similarly large dimensions. Perhaps of larger dimensions! Women are engaged in industry. Older men and women are found in industry than the men of military ages. Industrial risks exist, perhaps not so acute as the war risks, but of a very varied nature.

Before the war the psychiatrist was almost unheard-of in industry. Here and there some plant physician or accident specialist might be found who had had a partial training in nervous and mental diseases. But, if his

judgment was sharpened by that training, the fact was forgotten, and his success was no doubt laid to his "personality." Nowadays a few more men can be found associated with advanced plants. Yet in one plant system, where over 25 physicians held posts for one purpose or another, it transpired that no single one had ever had training in nervous and mental disease!

Nor can such men be easily obtained. He would be rash who should claim that there are 2,000 trained male or female American neuropsychiatrists. Probably there are far less than 1,000 able and willing to work in connection with industry. The majority of these are at work in other absorbing tasks. For some time it will be advisable for large plants to have part time consultants, chosen from amongst the more able mature neuropsychiatrists. Care must be exercised in the selection, for some of the professionally best of these men remain too analytic for the industrial situation and unable to see the values of rough and ready practical combinations which are the lot of employment managers and the minor executives. These consultants, if they once see the problem, can choose full-time younger medical aides, if such prove actually necessary.

As for (c),*psychiatric social work in industry,* we are entitled to expect large results therefrom. We have grounds of expectation not only from war work but also from the results obtained by psychiatric social workers long *ante bellum.*

Psychiatry Applied to Industry

Just as nobody would now think of denying the routine value of physicians and surgeons in industrial plants, so nobody can fail to note the good done by ordinary social workers in connection with industry. There is simply no dispute on either of these matters. To be sure, some managers may stress the *welfare* values of the doctor and the social worker, while other managers think of them as contributing to plant *efficiency.* But these are questions of the temperament of the managers, not of the nature of the results in the plants.

Now it requires no great refinement of viewpoint to see that, instead of a general practitioner of medicine, for some plant purposes (e.g., discharge, grievance, and certain turnover problems) a physician with psychiatric training would serve far better. The psychiatrist is by

training and experience a *specialist in grievances;* why is it not logical to apply this specialism to the grievances of industrial plants? On precisely the same grounds, the social worker with *psychiatric experience* is preferable to the general social worker for the purposes of industry, if we can prove that a considerable number of the more difficult plant problems are psychiatric or have a psychiatric tinge.

For the present argument, may I take for granted that the values of psychiatric social workers *outside of industry,* both in war work and in peace work, are generally admitted? To be sure, there may not be over 200 trained and experienced psychiatric social workers in the country at the present writing; accordingly it is only where they do exist or have been at work that their values are even understood, much less questioned. But there is, so far as I am aware, no dissentient word anywhere about the results of these workers, where they are in evidence at all.

But we have stronger evidence! For about five years the Psychopathic Hospital in Boston has carried on specific work in relation to the psychopathic employee. Some studies of the results of this work have already been published.[3] The facts of these psychopathic employees in and out of industry have been minutely followed by the psychiatric social workers of the Hospital, some of whom have specialized in the problems of employment. Probably more minute work has been done upon this problem from the hospital side than by any other agency. For a large part of the five years in question, a special committee has been interested in the matter. As an outgrowth of this work the Engineering Foundation, of New York, the joint research organization of the American societies of Civil, Mining, Mechanical and Electrical engineers, has under way preliminary investigations on the topic. We already know in specific instances what the psychopathic employee has cost the plant, the family and the state. We have cases under care which might have figured in dozens and scores of "turnover" analyses in different firms, had such analyses been made. In one instance five typewritten pages were

[3] I do not here refer to work on occupation neuroses and upon the nervous and mental sides of industrial accidents, which topics have also been the subject of special communications, but rather to the topic of the psychopathic employee in a more general sense.

occupied by the bare entries of employment and discharge by different firms. Of course, our work was primarily undertaken in the interest of the individual patient and secondarily in the interest of saving the public service additional expenditure for unemployed psychopaths. We did not at first have in mind the efficiency or welfare of industrial plants; but it has now become perfectly plain that the interest of the community broadly considered is identical with the interest of industry more narrowly considered and that both these interests are identical with the psychopath's own interest.

This is not the place to expound details of cases. In the Spring of 1919, we were able to demonstrate from the every day material of the Psychopathic Hospital clinic a number of things of great interest to employment managers sent to us for practical exercises. We showed man after man whom the employment managers acknowledged themselves capable of hiring on the spot for a variety of jobs. Yet the stories which the patients themselves readily and willingly unfolded upon very slight questioning were immediately indicative to the employment managers that the hiring of these applicants would have been a dangerous or delicate business, or a business requiring careful and special adjustments. We were able to show cases in which the special features brought out by the physicians or the social workers proved of decisive value in solving the patient's own problem of making a living and the industrialist's problems of securing a good workman. It is a well known and commonplace observation with employment managers and all who have to do with personnel that many a psychopath,—a cranky, grouchy, queer, or otherwise difficult person may be just the man wanted for a special task. An outline of these psychopathic hospital clinics as given in the Spring of 1919 to employment managers is being prepared for publication. The specifications for clinics of like nature for employment managers in the different great centers can readily be laid down, and some of these specifications will be shortly published.

Breadth of Mental Hygiene

The problem of mental hygiene is wider than medicine and wider than the branch of medicine that deals with nervous and mental diseases. The problem touches mental and social sciences and arts of the greatest breadth. Yet the indispensable core of the problem may well turn

out to be medical. I had the privilege, in the Spring of 1917, of many remarkable hours of consultation with the late Carleton Parker. He had, as everybody knows, come to a view of the great importance of the underlying ideas of mental disease and defect in the problem of industrial unrest. Every psychiatrist who appeared on the Pacific Coast was eagerly interviewed by Parker for what said psychiatrist might have to say on problems like those of temperament, monotony, fatigue and the like. It is a great wonder that an economist could have come independently to this point of view. Perhaps if more economists with thoroughly scientific training should live with the workmen as Carlton Parker did with the hoboes, the problem of hiring and firing, of promotion, of job selection, and in fact the entire problem of personnel, would get settled faster. Another economist, Prof. Irving Fisher, has gone so far as to use the phrase, industrial psychiatry, in published work.

Dr. Adler's Conclusions

There is a brief but interesting literature from the side of medical men themselves. The work on unemployment and personality at the Psychopathic Hospital as above mentioned, led to early publication by Herman M. Adler, M. D., now criminologist to the State of Illinois, but at the time of this work, chief of staff at the Psychopathic Hospital. On the basis of concrete case material, Adler rendered conclusions as follows:

"1. There are individuals in the community who, for a variety of reasons, are not able to regulate their conduct on the basis of experience. One of the difficulties that such individuals get into is unemployment. The results of their unemployment bring hardships on themselves and on their dependents.

"2. While some of these individuals show defects of such a severe nature that they may be regarded as hopeless and, therefore, can be segregated, there are others in whom the deviation from the normal is not sufficient to make them incapable of supporting themselves at all times and it is unwise to segregate them and prohibitively expensive.

"3. From our analysis it appears that there are two types of individual that experience these difficulties. One type, which is grouped under the headings of inadequate

and paranoid,[4] is afflicted with certain characteristics of personality which are not amenable to treatment. To maintain these people in the community it is necessary to modify the environment so far as possible in order to prevent, in the first place, the calling out of their peculiar reactions and, furthermore, to prevent their suffering the results of their acts; in other words, to keep a man "on the job" in spite of his personal unpopularity or inadequacy. The other type, grouped under the heading of emotionally unstable, suffers from the results of temperament. These individuals are subject to variations of temperament and the treatment of their unemployment must be guided by a knowledge of their tendencies so that environment on the one hand can be suitably influenced or chosen for them, and that the individuals themselves may be trained to counteract their impulses to some extent."

Miss Jarrett's Studies of Employees

Miss Mary C. Jarrett, now working on this topic under the Engineering Foundation, published briefly certain studies of the psychopathic employee as a result of her Psychopathic Hospital work. In general she found "employers quite willing to employ patients whose mental condition and industrial efficiency are frankly described and to retain them as long as they are able to do the work. Understood by their employers and taught to understand themselves, psychopathic individuals who would otherwise be thrown out of industry, may keep their places as efficient employees." She concluded by stating "that an important division of social psychiatry would be the application of psychiatric knowledge to industrial problems." She charted six cases in particular (one of dementia praecox,[5] one of depression of an al-

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[4] Paranoid, afflicted with delusions (or false beliefs) of persecution, e. g., by fellow-workmen, minor executives, relatives, in a variety of ways, e. g., damaging or hiding tools, non-promotion or raising of pay, nagging, poisoning.

[5] Dementia praecox, a non-fatal mental disease which in severe forms makes up the bulk of asylum populations, but which in milder forms is found in large numbers outside asylums. Dementia praecox has several types, e. g., a type of simple deterioration (as if the patient had acquired a degree of weakmindedness), a paranoid type (see footnote, p. 102), and a type, so-called catatonic, in which bizarre postures are maintained.

coholic, one of neurasthenia,[6] one of pronounced alcoholic psychosis, one of peculiar form of alcoholic jealousy and one of general paresis,[7]) which occurred respectively in a machinist, a tailor, a clothes presser, a packer, a teamster and another machinist—all of which cases, through the efforts of physicians and psychiatric social workers, became industrially adjusted. Alcoholism in some of this group of cases, may, of course, be regarded as one of the symptoms of a psychopathic constitution, exaggerating the original defect. It will be interesting to note whether national prohibition will abolish these problems. It is highly probable that many problems will not become abolished, but will be merely simplified.

In a later paper Miss Jarrett has discussed what she has termed shell-shock analogues under civilian conditions. She says concerning the war neuroses themselves: "The considerations that strike the psychiatric social worker in this situation are, first, the desire that this new, widespread knowledge of the neuroses that war is making prominent may be turned to the advantage and relief of civilians who suffer from similar troubles and receive inadequate consideration; second, that experience in the social care of civil cases of similar nature may be used to advantage in restoring soldiers suffering from shell-shock to normal social condition; third, that a thorough, intelligent public understanding of these disorders should be established against the day when the soldier who suffered shell-shock shall have again become a civilian, and the cause of his trouble may not be remembered acutely enough to arouse sympathy for symptoms that still persist."

She found that the analogues of shell-shock in civil life appeared frequently at the Psychopathic Hospital. The range of exciting causes was from trivial incidents, such as a quarrel or reprimand, to a profound shock, such as an accident in which the patient is severely in-

[6] Neurasthenia, a "nervous" rather than a "mental" disease (in the common acceptance of these terms), because victims almost never reach asylums. Sometimes known as the "American disease" or "nervous prostration." Instability, easy fatigability, "run-down" feelings, and complaints about supposed diseases of organs such as stomach, spinal cord, sex organs, are found.

[7] General Paresis, commonly termed "softening of the brain." All true cases of general paresis have been shown by modern work to be due to syphilis affecting the nervous system.

jured and a companion killed. She found another feature of the situation, which the layman cannot readil understand, namely, that the severity of the symptoms is not at all proportionate to the size or apparent importance of the cause. Treatment, however, must be relative to the gravity of the disease and not to the nature of the particular strain or shock which induced the condition. She narrates cases in detail to show first certain failures in social treatment which came about through lack of medical resources and inability to compel treatment, secondly, cases of pronounced success obtained by comparatively slight service, such as advice to the family or finding the patient a suitable position, and thirdly, cases in which results were only obtained with the most intensive social care.

These cases included a failure to cure a perfectly curable neurosis,[8] in an Italian laborer simply because medical facilities are not available in his home town and he cannot be brought to a central clinic; cases of character change following accident, cases of amnesia, and the like. Some of these cases might seem to run far afield from industry, but Miss Jarrett was able to find important connections between these cases and a variety of employment situations with the net result in many instances of complete adjustment. Something like half the cases of social work in mental hygiene clinics, such as that of the Psychopathic Hospital in Boston, will be found to throw light on various aspects of the employment problem.

Identification of Psychopaths

Readers of engineering journals are familiar with turnover analyses in which sizeable lists of the causes of discharge and unemployment are to be found. Jau Don Ball gives certain methods of examination which he has used, in his own phrase, "as scientific aids to industrial efficiency." It would be equally true to say that Ball's methods and those of others engaged in this work are also practical aids to industrial welfare. Efficiency ex-

[8] Neurosis, a term usually now restricted to the so-called functional neurosis, that is, a disease of the nervous system which is essentially and theoretically always curable. Modern writers almost always see a psychic element in these neuroses which include, for example, neurasthenia (see preceding footnote), psychasthenia (amongst which may be found conditions like the so-called "doubting folly" and obsessions), and hysteria (neuroses with various curable palsies, losses of sensation, etc.).

perts and welfare workers can unite in this mental hygiene program. Ball gives the following list of persons that might especially come under examination, *queer guys, eccentrics, disturbers, querulous persons, unreliable and unstable fellows, misfits, the irritable, the sullen, socially disgruntled, unsociable, negative, conscientious, litigious, bear-a-grudge, peculiar, glad-hand, gossipy, roving, restless, malicious, lying, swindling, sex pervert, false accusator, abnormal suggestibility* and *mental twist* types.

One might contrast this list of persons, who are either psychopaths or near-psychopaths or would obviously benefit by the kind of analysis which the psychiatrist would bring, with the following list of causes for removal from payroll quoted by Mr. Thomas T. Read, from a well-known department store blank form.

Other positions	1. Better salary.
	2. Former position.
	3. Going into business.
	4. More promising position.
	5. Position nearer home.
	6. To learn trade.
	Leaving city.
	To marry.
Health	On account of health.
	1. Own accord.
	2. Division superintendent's account.
	3. Doctor's orders
Dissatisfied	1. Did not like supervision.
	2. Distance too great.
	3. Refused temporary work.
	4. Refused to be transferred.
	5. Resented criticism.
	6. With salary.
	7. Did not like working conditions.
	8. Work too hard.
Unsatisfactory	1. Agitator.
	2. Carelessness.
	3. Dishonesty.
	4. Drinking.
	5. Fighting.
	6. Financial difficulties.
	7. Indifference.
	8. Insubordination.
	9. Irregular attendance.
	10. References.
	11. Superintendent's private file.
	12. Suspected of pilfering.
	13. Too slow.
	Reduction of force.
No reason	1. To go to school.
	2. To stay at home.
	3. Worked less than two weeks—failed to report.
	4. Worked more than two weeks—failed to report.

Among these orders the whole "dissatisfied" and most of the "unsatisfactory" groups are clearly subject to review from the point of view of mental hygiene. In particular may be mentioned as important to review from the standpoint of mental hygiene the "not liking of supervision," the "resentment of criticism," "too slow," "agitator" and even "dishonesty." There are 38 causes in the above list and fully half of them would give the mental hygienist pause. This is not to say that there are not plenty of dishonest people who are, so far as we know, merely delinquent and not psychopathic. There are, doubtless, plenty of instances in which the work assigned is actually too hard. There are also times, no doubt, when insubordination is a virtue and neither a vice nor a sign of mental disease. The point here made is simply that such causes as those above classified suggest review from a mental hygienic point of view.

Psycopaths and Strikes

Ball described the analysis of certain employees in a firm where two months after Ball's examination a strike occurred. Ball states that "in the case of every employee terminated for the group examined whether discharged or voluntarily leaving, the prediction of a possible abnormal conduct or a dissatisfaction was made in the laboratory report and recommendations to the employer." And further, "according to the records, everyone of the strikers had something wrong with them from a nervous or mental standpoint (nearly all having a psychopathic history); it was noted that with three exceptions the 'strikers' cited as agitators were among those grading the highest on the intelligence scale used." The intelligence scale used was a selection of tests made by Dr. A. W. Stearns during his naval work on the Pacific Coast, as examiner of recruits. Stearns promises early publication of his work, of which an advance account was given at a meeting of the National Association of Psychiatrists.

Of course no mental hygienist, least of all Drs. Ball and Stearns, would assert that all or many strikes could be prevented by advance studies of workmen. In fact Ball specifically says that "it could not be concluded from this or any other examination that all strikers, whether agitators or not,

are psychopaths; but this examination does show that the agitators in this group were the self-assertive ones and the ones grading the *highest in intelligence,* the others simply followed the leader. Nobody needs to say that there are not strikes having purely economic causes. Nobody needs to say that there are not strikes and other labor troubles due to mental disease or character defect either in the employment managers and minor executives or in the plant owners themselves. Some of the very conditions which make for self-assertiveness and success of a sort amongst labor leaders are conditions which make for the success of financial magnates and captains of industry. Nobody claims 100 per cent. efficiency for any of these or kindred proposals.

Practical Methods of Application

Ball draws several important conclusions. He thinks that what he calls medico-psychological laboratories (I think this term medico-psychological will hardly get across so well as the simpler and more comprehensive term, mental hygienic) ought to become principal departments of employment bureaus in large industrial organizations. Small plants might pool their interests for the establishment of such clearing houses. Labor organizations might well have, according to Ball's suggestion, representatives in these bureaus. Ball calls attention to the value of this work to the individual, to the industrial plant (emphasizing here the enormous reduction of labor turnover), to labor organizations (stabilization of personnel, clearer understanding of general problems), and to the community. He thinks the community would be aided:

(a) By lessening the number of undesirable floating population;

(b) By increasing the number of persons owning homes;

(c) By lessening the number of strikes;

(d) By decreasing the number of accidents and loss of life. (This especially applies to public service corporations. Instances are numerous where the lives of the public have been endangered by the *irresponsible acts* of morons,[9] epileptics, and mentally

[9] Moron, a term which has recently come into use for those feebleminded whose intelligence is higher than that of the imbecile or the idiot and roughly corresponds to the mental level of those above the age of seven, but subnormal.

and physically ill individuals, in the employ of public service corporations.)

I believe that to make this community program most effective we shall need to add to the laboratory for employment bureaus suggested by Ball also all the facilities and arrangements for medical social work and in particular for psychiatric social work. In short, we shall be compelled in the end, I believe, to add to the staffs of large industrial organizations mental hygiene clinics manned by competent practical psychologists and by psychiatric social workers. Very large or special plants or systems of plants might well require psychiatrists on full time, but all mental hygiene clinics for industrial organizations should have available the services of consultant psychiatrists at least.

In highly developed communities where the public service has developed effective stable mental hygiene clinics (such as the out-patient departments of psychopathic hospitals) large industries could send their special cases to these clinics for examination. Men who, for any reason might have to be removed from the payroll, would then fall into the most adequate hands for their further social care. Man after man would get adjusted in other and more suitable employment through the procedures of competent social workers, supported by analyses of temperament and fitness provided by the consulting psychiatrist.

In other situations it might prove a better plan for various firms to combine for the establishment of a common mental hygiene clinic. Mutual aid would be given to the organizations by this plan of combination and many a workman would soon find himself in a better job than he had ever had. As many contacts with the public service as possible should be made, since in this way some not altogether unreasonable suspicions by labor leaders will tend to be allayed. I have tried to indicate that the point of view of mental hygiene as applied to industry is neither a matter of efficiency alone nor a matter of welfare alone, but combines the two points of view.

Development of Public Health Work

The development of public health did not primarily

include mental hygiene. Public health work probably started as a rule in the arousal of the community's interest in sewage and simple sanitation problems. Next the public health specialists turned from these relatively simple engineering problems to bacteriology and the prevention of infectious diseases, at first through water supply and thereafter by a variety of measures. Latterly, however, we are witnessing the public health workers go over to special studies in personal hygiene. Studies of personal hygiene culminate in mental hygiene. The long, separate flow of work in mental hygiene will shortly fuse with that of public health in general.

The public health movement was for decades a movement for public bodily health. It must now become a movement for public health, bodily and mental. For example, the new school of industrial hygiene in Harvard University will pay important attention to the various problems of mental hygiene. *The Journal of Industrial Hygiene,* already in its first volume, contains reviews of mental hygienic interest. The United States Public Health Service has also developed alongside of its personnel for bodily health measures, the beginnings of a personnel for mental health measures. In the future, no doubt, every practitioner of medicine will get a grounding in mental hygiene. Parallel with this highly desirable state of affairs the rapidly growing group of public health nurses may, at some future time, get training in the principles of mental hygiene so that they may function in this field. Up to the present time, however, the number of physicians and the number of nurses, whether or not these physicians or nurses belong to the public health school, have not had the training or direction of interest such that they would prove very helpful in the mental hygiene of industry as here conceived.

Any clever layman can see some things with half an eye. The point of the movement for the mental hygiene of industry consists not in calling the attention of clever laymen to some obvious facts, but in suggesting to the clever layman that he look into the good work that has been recently accomplished in the various departments of mental hygiene, such as applied psychology, psychiatry and psychiatric social work. "Life," said Herbert Spencer, "is an ad-

justment of inner to outer relations." Most of the study of economists and political scientists has been spent on the outer relations. A good deal of new work has been done on inner relations, both normal and abnormal, by psychologists and physicians. Out of the philanthropy of the past has grown the humane but also efficient social work of the present day. The social workers must now do their best to aid in the adjustment of all these relations. This is a gigantic task; but is there anything to do but to go to it, to approach this task with all the expert light possible?

Summary and Conclusions

1. The general object of this paper is to set forth the existence and present rate of progress of a movement for the *mental hygiene* of industry.

2. This term *mental hygiene* is coming into general use to cover the expert activities of *psychiatrists* (*i. e.*, medical men interested in the problems of mental disease (including the mildest forms of temperamental deviation), *psychologists* (*i. e.*, scientific and theoretical experts, who are now turning attention to methods of mental testing designed to improve and replace the hit-or-miss methods of the past), and various non-professional or semi-professional *aides* (such as *social workers* with special experience in character-handicap cases).

3. The recent improvements in employment management and all activities dealing with industrial personnel show that industry is ready for the new movement and employment managers everywhere are displaying the keenest interest in the new ideas.

4. Meanwhile the war-time results of the experts in mental hygiene enumerated in paragraph 2 have given practical demonstration of the value of mental hygiene in a business partaking largely of the nature of industry, namely, the business of war.

5. The earlier literature of industry conclusively shows that the "mental hygiene of industry" is nothing new in its essence (witness, many older references to the *human element,* etc.), but today's contribution is the organization of older interests for a systematic attack on industrial personnel problems.

6. The keynote of this systematic attack on industrial personnel problems by means of mental hygienic data and methods is the pooling and coöperative combination of expert engineering interests and expert medical and

psychological and sociological interests: in brief, *the invoking by the expert in industrial personnel* of the aid of all available experts in personality, to the study of which the whole personnel problem must reduce.

7. The interested personnel man or lay reader is implored not to take sides for one or other claims or counter-claims by medical men, psychologists and others concerning the virtues of special methods. The topic is growing and a little controversial; but on the whole the quarrels about method are superficial and the unanimity of experts extraordinary (no doubt the trials of the war served to mature and season the experts on all sides).

8. Another warning! Every time the world has tried to measure things more accurately, many foolish persons have risen to protest. Not a few medical men and psychologists will rise to say over the same formula against the mental hygiene of industry. It is to be hoped that, at this late date of the world's history, we can jump this zone of senseless protest against what must inevitably succeed, namely, a program of more expert study of anything whatever, including the human personality, wherever at work.

9. The movement for a mental hygiene of industry is neither an outgrowth of the efficiency movement (Taylorism and the like), nor an outgrowth of the workmen's welfare movement (economic interest in shorter hours, better working conditions and the like), though mental hygiene does effectively combine "efficiency" and "welfare" (as it were, F. W. Taylor and Jane Addams).

10. On the contrary, a stream of independent developments in our knowledge of personality (medical, psychological, illustrated for example in the kind of insight into human nature displayed by William James) is now pouring itself into a branch of engineering—personnel management—which has been running parallel for some time. Let us think of the movement in the terms, not of F. W. Taylor nor of Jane Addams, but in terms of William James.

11. The text contains sundry definitions and general statements on these lines. Future papers will amplify the account.

12. Perhaps the argument for a mental hygiene of industry may be put in a nutshell form as a question:

Why should not industrial managers seek the aid of (a) those who can measure at least a few of our mental

capacities and have shown their abilities in the war work, of (b) those who are the best specialists we yet have in temperament and the best experts in grievances yet developed, and of (c) others less professionally trained who are capable of tracing out or helping to trace out the actual situation of (e. g.) labor "turnover" as shown in the individual instance?

13. In short, why not help to push on the movement for individualism in industry that everything sees coming and ardently hopes for?

APPENDIX

Notes Concerning Authors Quoted

Dr. Herman M. Adler, at present Criminologist to the State of Illinois and Director of the Juvenile Psychopathic Institute in Chicago, was formerly Chief of Staff at the Psychopathic Department of the Boston State Hospital and Assistant Professor of Psychiatry in the Harvard Medical School.

Dr. Jau Don Ball is Professor of Nervous and Mental Diseases in the Medical Department of the University of Southern California, Los Angeles, and has contributed various articles on nervous and mental diseases to medical journals.

Miss Mary C. Jarrett, A. B., was for many years associated with Children's Aid Society of Boston and was Chief of the Social Service at the Psychopathic Department of the Boston State Hospital from 1912 to 1918 inclusive and is now working under the auspices of Engineering Foundation.

Dr. A. Warren Stearns is Secretary of the Massachusetts Society for Mental Hygiene with offices in Boston, associated as consultant with the local Red Cross organization and with the teaching staff of Tufts College Medical School. He has contributed numerous articles upon psychiatric topics and was for years connected with the Danvers State Hospital and the Psychopathic Department of the Boston State Hospital.

The late Professor Carlton Parker was an economist and sociologist among the first to take up the psychiatric point of view in his topics (for his life and interests see An American Idyl, by Cornelia Stratton Parker, 1919).

Irving Fisher, Professor of Political Economy at Yale University, is well known for various public interests in economics and sociology and was largely instrumental in organizing the Life Extension Institute.

Notes on Certain Terms.

In the text of this article the distinction between psychologists and psychiatrists is clearly enough drawn. The psychologist in the past has been something of a theorist, and the so-called "brass instrument" psychology was a matter of college laboratories. At the best the results of these psychologists stood to industry, education and the arts as theoretical physics stands to railroad engineering. Even before the war, however, the terms, psychology and efficiency, fell into the habit

of being used together. The world was therefore not at all unprepared for such practical work as that of Lt.-Commander Raymond Dodge in certain naval problems, of Colonel W. D. Scott in the classification of army personnel and of Major Robert M. Yerkes in mental tests of recruits.

All this work by psychologists is distinct from the work of psychiatrists. Psychiatrists are medical practitioners in the field of mental diseases. Psychiatrists used to be termed "alienists," dealing as they were thought to do with alienations of mind. Psychiatrist is a better and broader term which has now come into general use and is rapidly supplanting the term alienist, which latter term might perhaps be reserved for medical men engaged in the medico-legal problem of responsibility of internment in asylums. It is important to insist that the psychiatrist now ranges over a much broader field than his colleague, the medico-legal alienist, or than anyone would have been ready to admit in the last century. His purview includes nowadays the slightest of character defects as well as the most serious of mental diseases.

Writing for employment managers and other laymen not skilled in the various mental sciences and arts, perhaps I should warn the investigator that the popular mind is inclined to talk of all these matters as psychology. Perhaps the term psychology does in some broad sense include all the mental sciences, but it is more than doubtful whether it ought to include or does include the various mental arts and types of technique (the so-called psycho technique of some writers). The practical thing for the investigating layman to bear in mind is that if he goes to the college professor of Psychology he may find somebody rather unsympathetic to mental tests and inclined to underrate their importance. If he consults an applied psychologist he may find an ardent mental tester who is not at all interested in what he thinks are the rough and ready, inexact conclusions concerning temperament, character, instincts and impulses which the less laboratory-minded man is willing to discuss. Now and then the investigating layman may meet an enthusiastic psychologist or physician prepared to explain the majority of difficulties on some special bases (e. g., the fear neuroses or sex). In the endeavor to make up his mind about these things, the peripatetic layman may run into sundry prejudices between psychologists and physicians or between certain groups of psychologists and physicians and certain other groups of psychologists and physicians. The layman must take heart. He must extract the honey of mental hygiene from a great variety of flowers each, no doubt, with what you might call a different honey index. And he must look out for charlatans.

I have occasionally used the term "neurologist" and even the term "neuropsychiatrist." There are certain differences between neurologists and psychiatrists and there is coming to be a group of men who, under the name of neuropsychiatrists, fulfill effectively both functions. From the standpoint of mental hygiene as applied to industry I have no doubt that men calling themselves neurologists are precisely as good mental hygienists as their brethren called psychiatrists. The neurologist is a medical practitioner dealing primarily with nervous

diseases. He is the sort of man naturally called in for a case of occupation neurosis (writer's cramp) and that sort of thing. The psychiatrist, or alienist,[10] as he was formerly called, is the sort of man who would be called in for a case of sudden outbreak of excitement; but the two fields so overlap that the distinction is likely to fall. As for the difference between certain diseases called nervous and some of the mild so-called mental diseases—well, it would puzzle the archangels to make a distinction. But although the layman need not particularly distinguish between neurology and psychiatry in his search for mental hygienic points, he must beware of thinking that he can get such points from any and every medical practitioner. He cannot.

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[10]Alienist, formerly the most frequent term for the specialist in mental diseases who was regarded as an expert who could tell whether a man was "alienated," that is, suitable for withdrawal from society.

LXIII

THE ENGINEERING FOUNDATION

TRADE-UNIONISM AND TEMPERAMENT

THE PSYCHIATRIC POINT OF VIEW IN INDUSTRY

By
E. E. SOUTHARD, M.D.

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"I look to no concrete results from those widely advertised industrial conferences held in our country in the latter part of 1919, simply because management and the engineering profession in all its branches were, in so far as I could make out, not properly represented. Capitalists who have once been engineers are capitalists notwithstanding. Labor leaders are prejudiced, and no doubt rightly so, for the practical purposes of their leadership. The public has interests that are diffuse rather than concrete and has no specialized knowledge either of financial systems and conditions of labor or of the theory and practice of management. But when the over conservatism of capital and the over radicalism of labor and the nebulous vagaries of the public shall have failed, as they will surely fail, to solve the industrial problem, then will be the time for engineers, in the broadest sense of that term, to be thrown into the game."

Reprinted from Industrial Management, April, 1920.

Trade-Unionism and Temperament

The Psychiatric Point of View in Industry

By E. E. Southard, M. D.

This is the second of Dr. Southard's *papers on "Mental Hygiene of Industry" resulting from the study supported by the Engineering Foundation. It was read as a professional paper at the Annual Meeting of the National Committee for Mental Hygiene on February 4 last. In a most interesting way the four types of trade unionism classified by Hoxie are compared and contrasted with the four classical temperaments. The suggestion is advanced that studies both by psychiatrists and psychologists will contribute much to our understanding of trade unionism, which may be a manifestation of mass psychology, and thus will assist the engineer, sociologist and statesman in their efforts to solve the problems of industrial unrest.*

Dr. E. E. Southard *was pathologist to the Massachusetts Commission on Mental Diseases, Director of the Boston Psychopathic Hospital, Director, Massachusetts State Psychiatric Institute.**—The Editors.

THE philosophy of trade unionism is yet to be written.

Its history is acknowledged to be baffling. Its present status deserves no other term than "hectic," and its future seems quite beyond prophecy. There are many trade unionisms rather than any single trade-unionism; but, whether one or several separate trade-unionisms, most observers would regard the trade unions as a phase rather than an end, as a technique rather than a purpose, sociologically considered. I was led to the present reflections while trying to clear the way for concrete work on the mental hygiene of industry.

Under the auspices of the Engineering Foundation some workers have recently been trying to discern whether the principles of mental hygiene could not be applied with some reasonable hope of success to the

* Dr. Southard died suddenly February 8

problems of industry. Now the tools of the mental hygiene movement, that is to say, the mental hygiene personnel as so far developed, fall into at least three groups, (a) a group of psychiatrists, (b) a group of psychologists, (c) a group of social workers; and in point of fact a few psychiatrists, a good many psychologists and a number of social workers with more or less mental hygienic training are already at work under various auspices in industry. It is not my task here to speak of the beginnings and progress of that work summed up recently in papers by Dr. Stanley Cobb, of the Harvard School of Industrial Medicine, entitled "Application of Psychiatry to Industrial Hygiene," and by myself, entitled "The Movement for a Mental Hygiene of Industry." In the present communication I wish to speak in a narrower sense (*i. e.,* excluding psychology in its technical sense) of *industrial psychiatry,* that is of the phychiatric division of mental hygiene and the possibility of its application to a leading problem of industry, namely, trade unionism.

Pooling of Psychiatry and Economics

"Industrial psychiatry" is itself no new topic. The phrase was perhaps first used by Irving Fisher, Professor of Political Economy in Yale University. But the idea that the psychiatric point of view might profitably be applied to industry has become almost a popular idea with the publication of "An American Idyl," the remarkable biography of the late Professor Carleton Parker, of California and later of the State of Washington, by his widow, who has carefully noted the progress of the psychiatric idea through Parker's independent thinking and his personal contacts with American psychiatrists. In short, there has been a much more sudden and productive pooling of psychiatry and economics than either mental hygienists or sociologists could have hoped.

But is such a union of mental hygienic and sociological interests as those foreshadowed by Carleton Parker likely to be early fruitful? After all, are we not dealing with pious hopes rather than with productive conclusions? I am afraid that many practical business men find little to hope for and much to fear in any collection of theoretical ideas that we may tie together with the name *"ology,"* or in any movement of the world dignified by the suffix *"ism."* Ologies and isms, the practical business man once might have risen to remark, have no

place in American practice—today he knows he should know better. The close relation of ology to ism in practice is well enough shown even in Marxian socialism. There seems to be no doubt that Marx got many of his ideas from the German philosopher, Hegel, and many others from the French social philosopher Saint Simon, and even some from the English economist, Adam Smith and the followers of Adam Smith. The comprehensive history of European thought written by J. T. Merz speaks of Marxism as a kind of materialistic paraphrase of the philosophy of Hegel and regards the materialistic results of Marx as working directly into the hands of the German historical school of political economy and jurisprudence. Herein socialism, a popular movement, has in turn influenced sociology, a theoretical science.†

A great many reasons, good and bad, have been given for the Great War, and there are even more predictions for the future of reconstruction than there have been explanations for the Great War. But whether we charge the receding terrors of the war or the febrile difficulties of the present to bad morals or faulty education of the people themselves or of their leaders, whether we regard the situation as good or ill in the hands of the slowly developing juristic system wavering between excessive social control and excessive individual liberty, or whether we throw the whole onus upon the shoulders of blind economic development—we would make a bad error if we left theory, science and philosophy out of the account.

The Banner of Theory

It has struck me that one of the nearest duties of Engineering Foundation, in entering work upon the mental

† That ologies can influence isms is shown in two other examples that may be borrowed from Merz's analysis. Fichte, in some ways the most German of philosophers, wrote a short tract in 1800 called "The Closed Industrial State," and in 1826 the independent, landed proprietor, Von Thunen, wrote a somewhat similar work entitled "The Isolated State with Respect to Agriculture and Economics" giving algebraical formulae for the natural wage of labor by eliminating rent. I commend to every reader the tenth chapter of Merz's work entitled "Of Society," especially any reader who doubts the relation of theory to practice in the development of the present human situation. Written as it was before the Great War (the fourth volume published in the year 1914), this work gives the most comprehensive brief account of the total situation which we now possess.

hygiene of industry, is to carry the banner of theory rather more proudly than it is sometimes carried. The engineer, especially the modern personnel manager, is no doubt most receptive to whatever his colleagues from other arts and sciences have to bring.

I look to no concrete results from those widely advertised industrial conferences held in our country in the latter part of 1919, simply because management and the engineering profession in all its branches were, in so far as I could make out, not properly represented. Capitalists who have once been engineers are capitalists notwithstanding. Labor leaders are prejudiced, and no doubt rightly so, for the practical purposes of their leadership. The public has interests that are diffuse rather than concrete and has no specialized knowledge either of financial systems and conditions of labor or of the theory and practice of management. But when the over-conservatism of capital and the over-radicalism of labor and the nebulous vagaries of the public shall have failed, as they will surely fail, to solve the industrial problem, then will be the time for engineers, in the broadest sense of that term, to be thrown into the game.

But expert engineers who "make the wheels go 'round" are not quite so authoritative when it comes to questions of the wheels themselves: Shall the wheels be turned at all? At what rate? and, especially, What new wheels shall be called to service? I spoke above of the causes of the Great War as possibly lodging in a faulty education, in evil morality, in a jural system falsely evolving, and in purely economic developments. The problems of reconstruction are likewise possibly mental, possibly moral, possibly incidental to adjustments of government and law, possibly under the influence of economic laws, old or new. It will be the duty of engineers facing industrial problems to deal in no narrow spirit or mere technique with those problems. The problem is beyond the grasp of scientific management except in some broad interpretation of that term which no one would be likely to accept. No mere education of expert managers, of workmen, of the public or even of the capitalists is likely to work, because the problem digs morally far deeper than in education within the grasp of the much vaunted modern publicity method. But although the problem is not one of scientific management, neither is it one to be solved bodily by the methods of charity and welfare.

Justice the Cry Today

Not happiness but justice is the cry today. The representatives of scientific management must come together with the representatives of social welfare. Those who stress the mental element must pool their proposals with those who stress the moral element. Both the efficient engineer and the expert social worker must bear in mind the cry for so-called social justice. Those who raise this cry seem always in some sense to rely upon improvements in government and law, whether those improvements are such as to make us have "faith in Massachusetts" or such as to hearten the legions of Bolshevism. The spokesmen of representative government in America or of the Soviet system in Russia claim with equal vivacity that they are out for social justice, and all intermediate types of politicians not only argue for social justice but would resent the statement that the principles of organized welfare work and, in the end, also the principles of scientific management, would not be successful at all alongside, and even by virtue of, the principles of social justice.

Thus we have advanced appreciably from the standpoint of Carl Marx. Carl Marx stood for self help, on the part of the scientists. There was no immutable principle of natural justice which would save the workmen. They would have to help themselves, as Lismondi cried: "What! is wealth then everything? are men absolutely nothing?" Marx's facts such as they were seemed to have been derived from Adam Smith, from Ricardo. The statisticians had built up a fabulous but harmless creation, "the average man." Ricardian economists then got up something which proved to be more of a Frankenstein, namely "the economic man." The theory of wealth upon its tripod of wealth, wages and profit, was the result of British economic thought. The economic man was a machine upon the Ricardian theory, and it was against this mechanical idea of the worker that Carlisle inveighed and Carl Marx leveled his suggestions for a complete overturn of the social order.

The Best Is Behind Scientific Management

I am inclined to see in the great movements for scientific management, for social welfare and for social justice the best efforts of the Head, the Heart and the Long Arm to solve the problem. Of course, we must not charge the Ricardians of old any more than a mem-

ber of the Taylor Society of today with delusions in human sympathy, and no doubt the movement for scientific management in its modern aspect has made much room for the moral motive, particularly in its study of the fatigue factor in industry. Again, it would not do to charge Carlisle of old or Jane Addams of the present day with irrationalism, or with a tendency to behead the system and run it by means of a heart only. As for the representatives of the Long Arm of the law, among the most liberal jurists whom I have had the pleasure of listening to, I fancy that none wants to acknowledge the desire either to behead the social animal or to tear out its heart. To be sure, if one took the principles of Austinian jurisprudence quite literally, the Long Arm and even the Big Stick are its most obvious forces.

Noble Endeavors Have Not Solved the Industrial Problem

You will rightly say that I have wandered far from the topic of trade unionism and temperament. My purpose in thus wandering was to show that a great series of noble endeavors has quite failed to solve the industrial problem. Some economists, both followers of Marx and ardent scorners, are still inclined to think that economics might well be left to solve its own problems as the blind were once commended to a system of representative leadership by other blind, who, let us hope, had strongly developed the sixth sense. But the great war and the problem of reconstruction do not argue strongly for the program to let things stew in their own juice. Neither the *laissez-faire* plan, nor the neatly rounded little Utopian systems of one or another economical theorist, practically works. Accordingly the Head, the Heart and the Long Arm of science tried their luck. Scientific management, social welfare, and social justice became by-words, each accomplishing something or a great deal according to the times and seasons. One great profession, that of medicine, had no share in these matters, unless very indirectly by influencing university teachers of men of science, moralists and lawyers. Today we see signs that medicine is to be called in. We cannot otherwise explain the numerous increases of interest in industrial medicine shown in more than one country and by more that one type of agency, official or voluntary, in our own country. Departments of hygiene are securing im-

portant contacts with industry, either solving the problems derived from the works, or carrying new laboratory results back to the plants themselves, or in a few instances laying down programs for aid in the personnel problem.

I want to make a plea for the inclusion in the program of industrial medicine of the neglected field of mental hygiene. I call attention to the fact that Engineering Foundation, representing the engineering profession, has taken up concrete beginnings of research upon this problem of the mental hygiene of industry, which will so develop that industry will shortly demand from the psychiatric branch of the medical profession various consultants who will not do their duty either by medicine or by psychiatry if they do not look attentively into these new matters. I think the ordinary physician. even the industrial physician, would look upon the topic of trade-unionism as very remote from his interest or knowledge. I am afraid that most mental hygienists would feel themselves wholly at a loss confronting trade-unionists. Nor will I make extravagant claims for mental hygiene or for its personnel. The problems which mental hygiene will attack are practical problems, and no practical problems are ever solved (so far at least as they deal with individual situations) *in camera.* Nevertheless, mental hygiene might have something to say in many problems.

Hoxie's Theory of Trade-Unionism Types

Let us take the late Professor Hoxie's work "Trade-Unionisms in the United States," published in 1917. Hoxie, according to his introducer, Dr. Downey, was originally trained in the "straitest sect of cloister economics," and was very able to sharpen a keenly analytical mind upon the subtleties of marginal utilitarianism. Hoxie spent more than ten years in intensive study of American trade unionism, which led him into various fields of inquiry, such as wage theory, socialism, pragmatic philosophy, social psychology, employers' associations, scientific management. His "Scientific Management and Labor" is well and favorably known. I want especially to speak of Hoxie's theory of the four functional types of Trade Unionism in America. To give some idea of Hoxie's methods, at the same time pointing out some relations of this work to mental hygiene, I propose to list a number of items taken from the report of Hoxie's students upon the trade union

program (embodied in appendix 2). From paragraph I, "Aims," may be quoted:

Expression of self, personality, temperament, group philosophy.

Higher intelligence and capacity for enjoyment.

Improvement of working conditions in health, exertion, independence, personal dignity, supervision and control.

Improvement of living conditions and standard of living; uplift of the working class; uplift of the community as a whole, self-help.

From paragraph II, "Principles and Theories," may be quoted:

Essence of social maladjustment is the wage system.

Low wages cause of most human ills.

Belief in the wage fund theory, or "lump of labor theory," causes opposition to industrial schools and immigration.

All workers are of the same benefit to society, whether skilled or unskilled, and all should therefore receive the same wages.

Competition between man and man is healthy, but between man and machine is injurious to man.

Society's obligation to the worker to help him obtain his rights, including the right to leisure, and right to education.

Organization is essential to freedom from oppression. Cheap workmen's hotels, minimum wage, etc., simply retard the one right way to better things—organization (Might is right when unionists win).

Right and justice are the rules of the game of the ruling class. Unions justified for the good they do, no matter how great the corresponding damage.

Ends justify the means.

From paragraph III, "General Policies," may be quoted:

Organization for mutual insurance.

No affiliation with welfare plans of other groups.

To act pragmatically.

Make use of:

Self-help only,
Strategic position,
Monopoly,
Strikes,
Boycott,
Violence if necessary,

Methods "within the law,"
Mediation,
Arbitration,
Conciliation.
To maintain efficient and high moral character.
To encourage industry, education.
Sabotage.
General strike.
Violence.
To discountenance violence.
To use any method in a pinch.
To educate and uplift union personnel.

From paragraph IV, "Demands," a few items may be quoted:

Equal pay for men and women.
No piece work.
Abolition of rushers, speeders.
No scientific management.
No change in class.
Protection against occupational diseases.
Sanitary shops.
Abolition of child labor, night labor.
Regulation of hiring, discharge, fining, docking, promotion.
Settlement of disputes.
Legislative demands.
Prevention of high-speed schemes.
Workmen's compensation.
Old age pensions.
Public as against private welfare plans.

From paragraph V, "Methods," may be quoted:

High moral requirement for membership.
Violence.
Intimidation of employers.
Scabs.
Sabotage.
Educational work through emotional appeals to public.
Education of public.
Inconvenience of public.
Social ostracism.
Mutual aid and insurance.
Grievance boards.
Moral suasion.
Control through superior competence and efficiency of union labor.

Practically the whole of paragraph VI, "Attitudes,"

might well be quoted since almost every item has some relation to mental hygiene.

Moral and industrial worth, not wealth, the standard of human greatness.

Physical power the motive force of everything; might is right.

The church and the State, the great pillars of capitalist society.

Contented workman is a pitiable object.

Those who kick without reason are better than those who do not kick at all.

Employers can meet with workers on a basis of justice to both, or on the other hand, "contracts with employers are not sacred."

Every welfare plan has a joker in it.

Conflict between materialistic bread-and-butter unionists and the idealistic members.

Trade-Unionism—the bulwark of capitalism.

A. F. of L. if not a labor organization, is simply a combination of job trusts.

When a man gets too wild for the A. F. of L. he goes to the I. W. W. so that the I. W. W. is a good thing.

A man is a scab when he gets in the way of your job, no matter how badly he needs the money.

Men's unions have bosses—women's do not.

Four Types of Trade Unionism

All the foregoing mass of even contradictory and various statements about unionism would form much grist to the psychiatric mill. The point of view of mental hygiene certainly needs to be applied to industry if such analyses of Hoxie's students are at all representative. The insight of Carleton Parker is certainly justifiable.

Perhaps Hoxie's most interesting contribution is his distinction of five function types, (four, if we exclude the group of so-called dependent trade unionism which relies upon the support of other forms of unionism, or is "yellow" in the sense of being created by employers). These four main types of unionism are:

Business unionism,
Uplift unionism,
Revolutionary unionism,
Predatory unionism.

Assuming that Hoxie's account of the functional types of trade unionism is approximately correct, so far as it goes, let us see whether the psychiatrist can

find any grist for his mill in an endeavor to learn what these functional types of trade unionism might mean in terms of the great fundamental psychic trends. Trade unionism looks like a phenomenon of mass psychology. No doubt the final account of trade unionism will be in terms of mass psychology, but at the present day we do not know too much about this so-called mass psychology. Moreover, it might be dangerous to apply modern and incomplete ideas of mass psychology to a social problem so red hot as the trade unionism of the present day.

Very near to the surface of the modern psychiatrist's consideration of any problem is the question of temperament. What temperament may mean in terms of mass psychology is, to say the least, doubtful. We can serve ourselves best with the distinctions of the psychology of the individual, simply because we have no mass psychology, in the matter of temperament. Granting that Hoxie was right in his definition of the functional types of trade-unionism, may we not profitably inquire how his results fit with what we know of temperament. I shall shortly be able to show that four main types of trade unionism discussed by Hoxie, correspond rather neatly with the classical types of temperament. However, my first point is not that trade unionists of one functional type are all temperamentally equipped in a certain way. In the second place, there is no question whatever that the labor leaders in a given way are necessarily men that would prove to be the vehicles of a particular temperament. Nor in the third place is it at all certain that the founders of particular trade union tendencies give pure examples of a temperament corresponding with their particular unionism. I am rather inclined to think that evidence will be forthcoming to prove the genesis of the different trade unionisms due in great measure to certain temperamental trends.

Four Types of Temperament

I do not trust to my own analysis of trade unionism, since, indeed, I have no special claim to training that would fit a man for such analysis. Likewise, I shall confine myself to the safe ground of a very ancient account of the temperaments. The psychiatrist cannot help having personal, and even partisan and political views of a topic like trade unionism. The psychiatrist like any other citizen, might therefore, import his own private views into the analysis. Without further pre-

face let us consider the classical temperaments as they have descended to us from Hippocrates and Galen. The "temperaments" of these Greek physicians were in the literal sense "humors," and good humor and ill humor have come down to us as results of Hippocratic and Galenical ideas. These men distinguished the following four types of temperament: The phlegmatic, the sanguine, the melancholic, and the choleric.

We distinguish sharply the power of a man's intake of sensory stimuli from his motor power of responding in various ways to these stimuli, present or past; but between intake of stimuli and discharge of responses man interposes his intellect and his emotions; the intaken stimuli are somehow combined in the mental processes termed intellectual (that is, inter-ligating). The behavior of man, that is, the shape his responses, muscular and glandular, take, is thus not merely a matter of his sensory intake of stimuli, but also a matter modified by memory, imagination and other intellectual combinations. However, besides sense, intellect and will (to use the old terms for these functions), we have also to deal with a man's emotions that may influence his behavior essentially and sometimes almost regardless of his sensory intake, his intellectual combining power and even the natural lines of his motor responses. There is an attitude of pleasure, of pain and perhaps of emotional indifference which modifies behavior. These are very inadequate words in which to describe what a man does as modified by what he takes in, mulls over, and has pleased or pained feelings about; but these over-simple words will serve for the moment to make my point, that sensory and motor power on the one hand and emotion on the other hand are apparently much more than the intellect. With these same senses, perhaps with the same muscles, the same glands, and no doubt with the same brain equipment, two men might act differently. We should be inclined to ascribe these differences to temperament.

Comparison of Trade-Unionism and Temperament Types

Thus, if we are analyzing trade unionisms from a temperamental viewpoint, we are not discussing how logically well or ill conceived these trade unionists are, that is, their intellectual value in the logical world; nor are we discussing their values in behavior, except as behavior is influenced by temperament. Let us now

show the four trade unionisms and the temperaments in parallel columns.

Classical temperaments	Functional trade unionisms
Phlegmatic	Business
Sanguine	Uplift
Melancholic	Revolutionary
Choleric	Predatory

There are obviously certain logical connections between the ideas conveyed by the terms for temperament and the terms for unionism. The phlegmatic temperament of relative indifference to pleasure or pain of ordinary degrees is precisely the every-day temperament of the majority not only of laboring men and all labor leaders but also of people in general. From business unionists in Hoxie's sense as from all persons with phlegmatic temperament we may expect business-like reactions, not too highly colored nor influenced by the extremes of temperament. In accordance with the warning previously expressed, I do not wish to say that business unionists may not now and again vivaciously, melancholically or vituperatively argue their points, but the logical machine of the business trade union in Hoxie's sense appears to be a machine in which vivacity, melancholy and irascibility are not effective forces. We do not regard the esteemed leaders of the American Federation of Labor as swayed by their different logical considerations, since we concede their proper partisanship for the men they represent.

As Hoxie sufficiently indicates, the uplift phenomena of unionism are still nigh universally displayed by the different types of craft and industrial unions. Perhaps there is no single actual union that expresses uplift, and nothing but uplift, in its work. Can we not safely conclude, however, that something like what underlies the sanguine temperament underlies the uplift movement? The modern psychiatrist would have to say concerning sanguine persons that they are often subjected to an opposite feeling, "the blues." Many such persons, technically called cyclothymic, belong by temperament to the uplifters. Whatever and whoever demands uplift gets the sympathy of these persons, whose interest may shift from week to week and month to month from one proper object of sympathy to another equally proper object. Perhaps we owe much of our effective social welfare work to the existence of the sanguine in the world.

Not even their blues, or what were originally called the blue devils, remain valueless, since on the upswing of their temperament they depict in glowing colors the terrible things felt by them when possessed of their devils.

Each of these temperaments, the phlegmatic and the sanguine, has its peculiar virtues. Each of us has felt each trend at different times. Is it not of practical social value to bear in mind the possibilities of these trends to evaluate not only other people's but one's own temperamental trends in this way?

Melancholic Temperament and Revolutionary Trade-Unionism

Perhaps the least obviously effective comparison here made is between the so-called melancholic, or atrabilious, temperament, and the revolutionary type of trade unionism. There is no neat correlation between black bile and the I. W. W. or the I. W. W.'s quasianarchistic forms of unionism. However, there is some suggestion of a parallel in the mental attitude of the revolutionary and that of the confirmed melancholic. The confirmed melancholic, particularly of the more advanced years, is apt to center thought upon certain ideas, which in frank cases of mental disease may amount to delusions. The point that we outsiders must bear in mind which might concern the revolutionary types of trade unionism, is not their emphasis on direct action, sabotage or violence, but the grounding of all their lives upon definite ideas or hypotheses. The emotional tone of this revolutionary unionism is almost always unpleasant. They are almost always in the state of felt passivity. The passivity they feel simply illustrates for them the passivity in which they conceive the world, especially the industrial world, to be.

If we approach the analysis of those revolutionary unionisms with the idea of their actual grounding in unpleasant violences and violences of felt passivity, we shall get on much better than if we try to interpret their behavior along simple lines of direct action. The direct action advocated by Sorel in his classical work on violence is a type of behavior grounded in an hypothesis philosophically held. We can best explain the direct action of Sorel on temperamental grounds, and this entirely aside from the logical accuracy of his conclusions. For aught we know, one

or more of the revolutionary types of trade unionism may be logically quite sound. Our one concern as psychiatrists would be to appraise correctly the share of temperament, in the total response or line of behavior taken by the revolutionary under examination, —e. g., by the philosophic syndicalist Sorel. Without stopping to inquire whether Hippocrates and Galen would concede our modern analysis of the melancholic temperament to be correct, let us concede that there does exist a type of revolutionary temperament of unpleasant feeling tone and of a felt passivity, quite capable of explaining many proposed revolutionary programmes.

Choleric Temperament and Predatory Trade-Unionism

Far easier is it to see the choleric temperament in the so-called predatory trade unionisms. Here are men working not upon the comparatively high intellectual levels of the revolutionary unionists, but upon lower, instinctive levels. The revolutionary and the predatory unionist may advocate and perform the same acts of violence and sabotage. The revolutionary will have his reasons,—the predatory will act on impulse; the revolutionary will have a predominating emotional tone of unpleasantness and will feel decidedly in the passive voice, like many a victim of out-and-out delusions. The correctness or accuracy of his belief makes no difference to his temperament. The felt passivity may be actual passivity or a fancied passivity. The effect upon the revolutionary's behavior is the same whether the felt passivity is real or imaginary. On the other hand the predatory unionist may well feel himself frankly and gloriously in the active voice. His emotional tone may be unpleasant enough, though in the midst of anger or serene pleasure. However, the predatory unionist, like any impulsive predatory person, is not a very pleasant fellow on the whole, either taken from the inside or from the outside.

I do not know how sound these parallels between the ancient temperaments and the modern unionism types may be. Upon some such lines, however, I am convinced that we shall learn to distinguish not only the functional types of trade unionism, but also other types that function in the modern world. We are very far removed from the Average Man of the French statisticians and an Economic Man of Ricardo

and his abysmal failure. The world looked for many years for statistical resemblance amongst men. We should now look for qualitative differences. If it should turn out that Hoxie has made a fundamentally accurate study of the types of trade unionism, the analytic point of view of modern psychiatry may be of considerable help in the further study of these trade unionisms. The psychiatrist may not be sure that Hippocrates and Galen were more than approximately correct in their account of the temperaments, but he may be able to add a little here and a little there to the classical doctrine, or he may be able to overthrow the classical distinctions altogether. Upon some such analytic line shall he be able to help the world in its confrontation of many problems.

Mental Hygiene Promises Help in These Problems

It will not turn out to be a matter of the Head alone, that is of a particular logical and scientific evaluation of the proposed system. It will not turn out to be a matter of the Heart alone, that is a matter of social welfare, rose-tinted or morocco-bordered by the temperaments of uplifters. It will not be a matter of the Long Arm of the law until the law, so to say, can tell its left hand from its right, can distinguish individuals one from another more than its general relations now permit. Mass psychology and mass psychiatry may be in the future of undreamed proportions and quality. We have only the minds normal and abnormal of the individual man to go upon. Can we discern in the nebulous and mobile outlines of trade unionism once more recurrent, the classical trends of temperament? If we can be sure of our analysis here, we can no doubt meet the problems of trade unionism with much more understanding and with very much more sympathy.

Mental hygiene, I venture to say, as represented both by psychiatrists and by psychologists, will make in the long run a considerable contribution to sociology. Out there we speak in terms, more of dreams than in performance, but for that matter the trade unionisms are themselves no better off in this respect, nor has the Head with its scientific management, the Heart with its welfare programme and the Long Arm of the law in its ideal of social justice, given us much more than promise.

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LXIV

THE ENGINEERING FOUNDATION

THE MODERN SPECIALIST IN UNREST

A PLACE FOR THE PSYCHIATRIST IN INDUSTRY

By
E. E. SOUTHARD, M.D.

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The Modern Specialist in Unrest

Place of the Psychiatrist in Industry

By E. E. SOUTHARD, M. D.*

This is the third of Dr. Southard's *papers on "Mental Hygiene in Industry" resulting from the studies supported by the Engineering Foundation. It was read at the Fortieth Anniversary of the Boston Society of Psychiatry and Neurology, January* 15, 1920. *It develops the thought that the trained psychiatrist should become the consultant of industry, particularly in regard to those matters which tend to increase or lessen the feelings of unrest in individuals which probably are the starting point of group unrest or, as we commonly say, industrial unrest. Thus the work for such an industrial psychiatrist would be both curative and preventative.*

Dr. E. E. Southard *was pathologist to the Massachusetts Commission on Mental Diseases, Director of the Boston Psychopathic Hospital, Director, Massachusetts State Psychiatric Institute. His preceding papers appeared in the February and April issues.*—THE EDITORS.

INDUSTRIAL medicine exists. Industrial psychiatry ought to exist. That industrial medicine exists is attested by the founding of national and local societies, journals, personnel groups and the pursuit of researches. Industrial psychiatry, while it has logical claims to existence, has hardly taken shape. In a paper on "The Movement for a Mental Hygiene of Industry"[1] I have collected those few references which indicate the probable future course of the industrial psychiatry, of industrial psychology and of the new field of psychiatric social work as applied to industry. In that communication on the general aspects of the new movement I tried to state the issues for non-medical readers especially for those advanced engineers, employment managers, and other industrialists who see more in industry than either its "efficiency" aspect narrowly taken,

* Dr. Southard died on February 8.

[1] *Mental Hygiene,* January, 1920, and INDUSTRIAL MANAGEMENT, February, 1920.

or its "welfare" aspect narrowly taken. I would be pleased if I could in the present communication awaken the interest of psychiatrists themselves to what must be conceived as another immediate addition to the community functions of the psychiatrists. In some sense then the present communication is a foil to my earlier paper.

I seize the opportunity afforded by the fortieth anniversary of the founding of the Boston Society of Psychiatry and Neurology for the present purpose, because that society is well representative of the two sides of psychiatry that have developed rather independently from (a) the necessities of the state care program for the insane on the one hand and (b) from the necessities of private psychiatric and neurological (including medico-legal) practice. Your Society, made up as it is of both kinds of psychiatric practitioner, public and private, ought to be especially sound upon new matters like the development of industrial psychiatry touching as it must public, social and individual interests alike. As my hearers are thoroughly aware, between (a) the work of the public psychiatrists, whether busied with hospital administration and treatment or concerned with medico-legal decisions and (b) the work of the private practitioner in neurology or psychiatry busied with the individual problems of diseases for the most part falling short of the asylum degree, there has come recently to fairly complete logical development the new field of social psychiatry—a field wherein the problems of the probate court and the problems of the consulting office are amplified and developed by a hundred ramifications in the social web.

Value of Psychiatric Worker

Many of our public practitioners of psychiatry, that is, the institution men of the Commonwealth of Massachusetts, are already convinced of the values of psychiatric social work in this new intermediary field, lying between public practice and private practice in mental diseases as these fields have been construed up to recent times. The files of the Psychopathic Hospital will soon contain many thousand socially investigated cases, derived from Boston and the surrounding metropolitan district. But private practitioners in neurology and psychiatry are also becoming aware of the values of the psychiatric social worker for any private practitioner who takes his job seriously and seeks to solve his psychiatric problems with all modern aids. The three fields of public, social and individual practice in psychiatry are thus well logically in mind in this

Commonwealth. Indeed they are becoming clear to most other Atlantic Seaboard states and to all urbanized communities that have faithfully undertaken the work of mental hygiene on approved lines.

Yet some of us (I fear) may still regard this intermediary field of social psychiatry as more a theory than a condition. Luckily the men who think practice more important than theory are fast dying out or undergoing belated conversion through reflection on the successes of theory in the Great War. If, however, you actually do meet one of these incorrigible practical men who will see nothing in theory, it is as a rule enough to show him the results of social psychiatric practice amongst the relatives of the victims of neurosyphilis (general paresis and the like) to convince him that mental hygiene has unlocked a brilliant and efficacious novelty for public health in its work upon the so-called "syphilis of the innocent." I make this point about social psychiatric practice somewhat at length, not so much for my present hearers as for others who may read the printed remarks and wonder whether after all there exists a personnel to attack such widely ramifying problems as those of industrial psychiatry. There does exist the appropriate personnel for work in industrial psychiatry. There is, to be sure, not enough such personnel; but there do exist competent workers who can be multiplied as soon as industrialists begin to crave this personnel and as soon as psychiatrists see the peculiar values of the new work.

Why Mental Hygiene of Industry

Why do I speak of the mental hygiene of industry? Why should the medical man enter fields like those of psychology and social work, fields in which he is not competent by special training or daily experience? We are all aware that there is a borderline between the work of psychiatrists and the work of psychologists and that claims and counterclaims have been made by representatives of psychiatry and psychology. Why is it not better for psychiatrists to pursue their own expert ways, leaving psychologists to theirs? We are all aware that heated discussions have taken place in national associations, anent, e. g., the so-called "diagnosis of feeble-mindedness," which seems a medical problem to the psychiatrists and an educational problem to the psychologists. It seems to me, however, that although heated discussions upon abstract lines may be got up at society meetings, there is absolutely no practical or concrete difficulty in marking out the peculiar use of the psychiatrists and the psychologists

in a particular concrete case of mental disease or defect. For example, at the Psychopathic Hospital the work of Prof. R. M. Yerkes, psychologist to the hospital, ran with perfect smoothness alongside of the work of the psychiatrists in hospital wards and the out-patient service. On these practical grounds, as well as on sound theoretical grounds, I conceive that it is both tactical and strategic to place psychiatric art and psychological science under the one head of mental hygiene; the term "mental hygiene" has medical suggestions, but it also has equally pronounced suggestions of normality and health. Where so many of our problems in the social division of psychiatric practice lie along this borderline between normal and abnormal, I conceive that the term "mental hygiene" is perhaps the best that can be found to describe the sudden enlargement of the psychiatric range in recent years. But, together with the psychiatrist and the psychologist, I would also range the psychiatric social worker as a third kind of mental hygienist. The mental hygiene of industry will, of course, require the services of all three types of mental hygienist, as I endeavored to make plain in my communication for laymen entitled "The Movement for a Mental Hygiene of Industry." As psychiatrists and physicians we shall not forget the importance to mental hygience in general, of mental hospital nurses, of occupation workers, and specialized types of teachers for mental diseases and defects. But these latter varieties of mental hygienist are not so much in point in the primary field of industrial psychiatry. In that field a working party, composed of psychiatrist, psychologist, and psychiatric social worker should if possible have added thereto a person skilled in tabulation and statistics.

I have just employed the phrase *working party* in the mental hygiene of industry. Such a working party would be of value in almost all other fields of mental hygiene, for example, in the survey of a state or district, an occupation group, a racial group, or any other special group of persons whose mental hygiene demands attention. Parenthetically, I am sure you will all agree that there is hardly any group of persons in the world that would not benefit from mental hygienic analyses made upon the triple lines herein indicated. Thus such a working-party composed of psychiatrist, psychologist and social worker can already be found in advanced juvenile courts, and even in certain courts for adult cases, and would undoubtedly be of the utmost service in all domestic relations courts. Again, in schools, in various institutions for the care of chil-

dren this combined insight would penetrate many a dark corner.

Industry Today's Nearest Problem

But industry seems to me to be the nearest problem today to the hands of mental hygiene. One is impressed with the readiness of industry for such working parties in mental hygiene. The war has brought industrial problems into sharp relief. Reconstruction has altered the focus in places but has not abolished the problem. Above all there is at the present day the so-called industrial unrest, a problem met apparently with not too great intelligence, if we can judge by the nullities and silent dispersal of certain national industrial conferences in our country. To be sure, the Royal Commission on Industrial Unrest in England during the war time (1917) did important service in laying down certain concrete findings and recommendations, but these results were war-time results confined to Great Britain. The psychiatric reader of the Royal Commission reports must be convinced that greater and more significant results could have been obtained if the principle of what I have called the mental hygienic working-party could have been adopted in the British investigation.

A Mental Hygienic Working Party

A word or two about respective functions of the members of a mental hygiene working-party. For the benefit of those who come to this problem for the first time, let me insist that such a working-party is not proposed for the purpose of supplanting the employment or personnel manager or any other major or minor executive in the industrial plant. I hope to convey by the term "working-party" the idea of an investigation occasional rather than permanent, carried out by special officers having the weight of certain connections outside of the industrial plants themselves. Of course the psychological examiner will, no doubt, prove a relatively permanent portion of the organization of an industrial plant, as soon as the managers of these plants get clearly in mind the army successes of psychology in the classification of personnel and the elimination of the unfit through group and individual mental tests. I think that portion of the propaganda for mental hygiene may be regarded as properly under way. I am bound to say that I think the plant has been adopting it more as an efficiency device than as a welfare or social adaptive measure, at least in certain plants. But this tendency to exploit the values of

mental measurement for the mere elimination of the individual from a particular plant will be short-lived if we can somehow kindle the spirit of mental hygiene in the whole industrial problem. After all the psychological examiner will find himself of greatest value in the employment or hiring side of the plant's work. As the years pass, the psychologist may also learn to contribute to the problem of promotion upon lines of vocational psychology. But for the present the psychological examiner, in the narrow sense of this term, will be of decidedly lesser value in the interpretation of the discharge-rate or turnover in industrial plants. The industrial plant should have the list of discharged employees gone over from time to time by a consultant psychiatrist or a neurologist interested in the psychiatric side of this work. Such consultant phychiatrist should be in complete touch with the psychological examiner and should have at his disposal such records of mental testings or other recorded impressions as the psychologist may have. He should likewise be able to secure and interpret the records of social work, especially these made in connection with discharged employees.

The psychiatric social worker, like the psychological examiner, will probably become a permanent element in the plant, although most of her work may well be done outside its walls in the community, and especially in the families even of those who become industrially disabled and of those who are discharged, for such reasons as are consistent with the spending of the plant's time and money on their families. Luckily, in progressive urban communities the standard of social welfare has been advanced at least to the point reached by the standards of efficiency inside the plants. The result is that by a minimum of exertion, on the part of the social worker proper, transfers of these families of discharged workmen to other agencies can be arranged for. Where there are a number of industrial plants in a single community, the social workers connected with these plants would naturally be closely associated with one another in their social conferences and society meetings. Industrialists tell us that coöperation is more the order of the industrial day than competition. However this may be in industry as a whole, there can be no doubt that the social treatment of families of employees discharged from plant A will benefit the turnover sheet of plants B and C, etc. If plants B and D employ social workers of like skill, plant A will in turn benefit in its turnover sheet. In brief the welfare of the discharged means the effici-

ency of the plant. The general problem of turnover is aided by the well-known principle of mutual "back-scratching." Meantime the welfare values obtained for the community as a whole run beyond the superficial relief of the industrial skin.

Combination of Medicine and Engineering

How soon it will be possible to make the physician in general and the medical social worker not especially trained or expert in mental problems see eye to eye with the psychiatrist and the psychiatric social worker in this matter of the mental hygiene of industry, is hard to say. However, it would appear from the operations of national and local societies of industrial medicine that physicians in general are becoming much alive to the virtue of this new combination of medicine and engineering. Psychiatry has made such strides in relation to the more superficial problems of social work that psychiatrists are often overwhelmed with the kind and degree of expectation uttered by social workers. The extreme range of expectation is shown in the files of the out-patient department in the social service of the Psychopathic Hospital in Boston. Much is expected of the psychiatrist in the new social division of his practice.

It is particularly in the grievances that come to the attention of the employment manager that the psychiatrist will find his work laid out. The following entries will readily suggest to the psychiatrist what sort of investigation ought to be carried out, especially with the aid of the psychiatric social worker:

Certain causes of removal from payroll:

- Did not like supervision
- Refused to be transferred
- Resented criticisms
- Did not like working conditions
- Work too hard
- Agitator
- Carelessness
- Dishonesty
- Drinking
- Fighting
- Indifference
- Insubordination
- Too slow.

There is also a paragraph called "Superintendent's private file" among the "unsatisfactory" groups of removals that might well be looked into by the consulting psychiatrists. Where do all these grudge-bearers, agitators, drinkers, fighters, and lazy persons go? Some

of them figure in the discharge files and turnover analyses of not distant plants within a comparatively short time. We may talk of the solution of such problems as a duty of the community; but it should not be long before industrial plants themselves recognize the efficiency and welfare virtues of attending as strictly to their human outgo as to their human intake. I mentioned the work of the British Royal Commission on industrial unrest in 1917. I present a summary of their findings made by the Right Honorable G. N. Barnes, M. P., not because all of the 14 items are particularly related to our own or any special problem in industrial hygiene, but to show the general nature of the Royal Commission's work.

Summary of the Industrial Unrest Findings in England, 1917, by G. N. Barnes, M. P.

1. High food prices in relation to wages, and unequal distribution of food.
2. Restriction of personal freedom and, in particular, the effects of the munitions of war acts. Workmen have been tied up to particular factories and have been unable to obtain wages in relation to their skill. In many cases the skilled man's wage is less than the wage of the unskilled; too much centralization in London is reported.
3. Lack of confidence in the Government. This is due to the surrender of Trade Union customs and the feelings that promises as regards their restoration will not be kept. It has been emphasized by the omission to record changes of working conditions under schedule LL, article 7 of the Munitions of War Act.
4. Delay in the settlement of disputes. In some instances ten weeks have elapsed without a settlement, and after a strike has taken place, the matter has been put right within a few days.
5. Operation of the military service acts.
6. Lack of housing in certain areas.
7. Restrictions on liquor—this is marked in some areas.
8. Industrial fatigue.
9. Lack of proper organization amongst the unions.
10. Lack of commercial sense—this is noticeable in South Wales, where there has been a break away from faith in Parliamentary representation.
11. Inconsiderate treatment of women, whose wages are sometimes as low as 13£.
12. Delay in granting promises to soldiers, especially those in class "W" reserve.

13. Raising the limit of income tax exemption.
14. The Workmen's Compensation Act—the maximum of 1£ weekly is now inadequate.

Among the recommendations of the British Commissioners are to be found recommendations concerning:

1. Food prices (of which the commission stated there should be an immediate reduction with an increase price partly borne by the government and with a better system of distribution).
2. Industrial councils on the principles of the Whitley report.
3. Authoritative statements by the government on further increase of output (war-time conditions).
4. Participation by labor in the affairs of the community as partners rather than as servants.
5. Publicity in certain matters relative to leaving employment.
6. Publicity by the government concerning its pledges already given.
7. Raising of the 1£—maximum under the Workmen's Compensation Act.
8. Announcement of the policy as regards housing.
9. Skilled supervisors to receive bonus.
10. Closer contact to be between employer and employee.
11. Pensions Committee to be granted more discretion in treatment of men discharged from the army.
12. Certain agriculturists' wages to be raised.
13. Colored labor not to be employed in ports.
14. A higher taxation of wealth (by one commissioner).

Causes of Unrest in England

As for universal causes for unrest in England there were four, according to the Commission's report:

1. Food prices and distribution of supplies.
2. Restriction of personal freedom.
3. Card system for military and industrial service.
4. Coördination of government papers.

Certain acute though not universal causes of unrest were:

Housing
Drinking
Fatigue.

The Commission also speaks of "psychological" conditions and remarks that "the great majority of the cases of industrial unrest specfied in the (8) district reports have their root in certain psychological conditions."

Among these may be mentioned:

Lack of confidence in the government.
Feeling of inequality of sacrifice in army and industry.
The idea that solemn pledges were broken and turned into "scraps of paper."
Feeling of unreliability of certain trade union officials.
Feeling of the uncertainty of the whole industrial future.

The Commission was no doubt justified in laying enormous emphasis on what is called "psychological" conditions. The psychiatrists and the medical men in general must feel that the blanket term "phychological condition" covers a good many psychiatric difficulties. Thus, whoever follows the strong trend to individualization in medicine, psychiatry, in education, both intellectual and moral, and even into the law courts, must be convinced that individualization should proceed to greater lengths in industry. There is nothing more wide-spread in modern sociology than certain ideas about group action as the be-all and end-all of progress and failure in social developments. As one author puts it, group experience leads to group thought, group thought to group action. If we take, for example, the universal causes of unrest summarized by Barnes, of England, we shall, of course, be convinced that food prices might well be a group experience, a poor distribution of supplies might be to a large extent a group experience. There would also be a group experience of the evils of card systems which might lead to group thought and certain unrest of mind might create tendencies to strikes, and disturbance would tend to follow group experience and thought as to prices and service cards. When it comes, however, to a question of the restriction of personal freedom and to a question of temperamental incoördination, it might be observed that these are hardly group experiences as much as individual experiences. The workman who objects to being automatically passed from one sphere of labor to another may not make himself heard effectively in group thought, so with the victim of some incoördination on the part of government departments. But it certainly must be true that the effects of such restriction of freedom and temperamental incoördination are, as a rule, individual. The voices of those victims are raised alongside of the voices of general unrest concerning food prices and the service-card system.

Reconstruction of Personal Freedom

We cannot help thinking that the principles of social work, and especially psychiatric social work, applied to the problem of the reconstruction of personal free-

dom or temperamental coördination will solve most of the problems. The matter of automatic transfer from certain spheres of labor is, of course, a war rather than a peace matter, but the item will serve as well as another to indicate that universal causes of unrest need not be the product of group experience, need not have led to group thought, and need not lead to group action unless in the presence of other general causes of unrest. Many of these problems, possibly the majority of them, are extraordinary rather than main problems. The same holds for the "acute" and not the "universal" causes of unrest, most of which are described by the commissioners as arising logically from different family housing, drinking or fatigue problems, even when it comes to such a problem as that of lack of confidence in the government specified amongst the findings as No. 10, "lack of commercial sense."

We find from the commission's report that this lack of commercial sense was especially noticeable in South Wales where there had been a break away in faith in parliamentary representation. Of course I cannot know any single important fact relative to South Wales and its break away from the democratic faith, but certainly here is a local condition which no doubt had local causes some of which are almost certain to have been due to the operations of a particular man or group of men.

Psychiatrist Should Know How Unrest Will Run

This introduces us to the most general aspect of the unrest problem, the aspect which led me to give the somewhat cryptic title to my communication of "The Modern Specialist in Unrest." It may be (or as I suspect it may not be) that group experience leads to group thought, group thought to group action as the ordinary course of events in social developments. But whether these developments are group matters or not, it remains true that most of the information which we possess concerning group psychology and group psychopathy is derived from the psychology or the psychopathy of the individual. If this statement be accounted true, then I do not need to insist that the psychiatrist is rather more likely than any other expert to know how the main lines of unrest will run. Unrest on the part of the individual is the big problem of the psychiatrist—year in and year out he comes in contact with the finest, as it were, and the most brilliant examples of unrest, namely, certain patients in his wards. If this general account of things be correct, the psychiatrist

ought to have a message for industry. Psychiatric knowledge about the unrest of the individual ought to be turned to account in our analyses of group unrest.

The main thesis of the present communication is that a psychiatrist has a place in industry. I think that he will have a place in the routine of industrial management, not as a permanent staff member (save in the instances of very large firms and business systems) but as a consultant at stated periods relative to the matter of grievances, complaints and dissatisfactions, actual and potential. The function of this occasional consultant would be preventive rather than curative of the general conditions of unrest. How far can we think of the industrial psychiatrist as not merely a preventive agency, but as a curative agency for conditions of unrest? The future must decide this question.

What is unrest? The theory that group experiences leads to group thought, which in turn leads to group action, may be sound theory for a portion of industrial phenomena, but individual experience, individual thought, and even individual action are also factors in industrial situations. How far is unrest a matter of group, or crowd or mass psychology? How far does mass psychology depend upon the psychology of the individual member? It will not be wise to generalize to the effect either that industrial unrest is entirely a group phenomenon or that it takes its rise entirely in the minds or hearts of individuals. We have seen that some of the causes of unrest in England might well be matters of group psychology, but that other causes of unrest seem almost in their nature to have been of individual origin.

Individual Aspects of Unrest Problem

That portion of the unrest problem which depends not upon group experience, but upon individual experience, not upon group thought but upon individual thought, and finally not upon group action but upon individual action, is the proper topic for the psychiatrist. The psychiatrist has always been a specialist in unrest;—unrest, to be sure, confined within asylum walls, particularly. In company with the psychiatric social worker, the modern psychiatrist has under more or less definite supervision, large numbers of the so-called psychopathic personalities, being persons who are not insane in kind or degree to warrant their commitment to institutions, but who are psychopathic enough or in such wise as to benefit from community supervision. It is this modern contact with the psychopathic personalities, or instances of so-called psychopathic in-

feriority, or psychopathic states that makes the modern psychiatrist a specialist in a kind of unrest that interests the community very deeply. These psychopathic personalities have been even recognized in the immigration laws and in the official tabulations of the Army and Navy under the terms "Constitutional Psychopathic Inferiority," "Constitutional Psychopathic State," and similar designations.

Contribution of Engineering Foundation

It is important for the modern psychiatrist not to "hide his light under a bushel," he must step forth to new community duties. It is on this account that I conceive that a recent step of Engineering Foundation is of so much importance. Amongst the earliest problems undertaken by Engineering Foundation is the problem of mental hygiene of industry. To the writer, the Director of the Massachusetts State Psychiatric Institute, was entrusted a research of definite dimensions, relative to the mental hygiene of industry and the problem of mental abnormalities in relation to industrial personnel. The enlightened officers of Engineering Foundation immediately found the ramifications in the research of mental hygiene of industry so numerous and broad that a plan is being mooted for investigation of the entire problem of industrial personnel. On the suggestion of Engineering Foundation, National Research Council appointed a committee composed of representatives of its Divisions of Anthropology and Psychology, Educational Relations, Engineering, Medical Sciences and Research Extension, to consider the scope of investigation into industrial personnel.

It seems to me that as psychiatrists we should help this movement wherever it becomes practically possible. The practical possibilities of helping lie in connection with the fact that the majority of our male patients have either come out of industry or are going back into industry in some capacity. Special investigations of the individual patients with respect to their industrial status and future should be made. The information which the psychiatrist possesses concerning personality, temperament and special abilities, as modified by mild mental disease and defects, should be at the call of the employment manager. There should be a drawing together of the psychiatric and industrial interests of all communities. The psychiatric social workers of the state institutions will meet similar workers from the industrial plants to discuss the individual fates of particular discharged workmen. Psy-

chopathic persons can be fitted into industry far more successfully than most psychiatrists and industrialists are feign to believe. The employment work at the Psychopathic Hospital during the last four years has definitely shown these adaptations of the psychopathic employees to be both numerous and effective.

I do not need to rehearse the early conclusions of Dr. Adler concerning unemployment personalities, based upon Psychopathic Hospital studies, more than to recall the immediate sub-division which he made into

(a) Feebleminded
(b) Cyclothymics
(c) Paranoiacs.

Every employment manager is aware of the existence of the feeble-minded and of their availability for certain kinds of work. Industrialists are also quick to recognize the cyclothymics with their ups and downs of emotional mood as actual inhabitants of mills and mines, and much can be quickly taught the employment manager concerning the special virtues and faults of these victims of the cyclothymic constitution, and even of the more severe forms of manic depressive psychosis. The paranoiac patient is likewise ready to be recognized by the employment manager as a man with a grudge or chip on his shoulder. In fact, the task of letting in a little psychiatric light upon these problems is not so difficult as might be conceived; the success of Psychopathic Hosiptal clinics for employment managers in the summer of 1919 attests the value of spreading these practical doctrines of mental hygiene among industrialists.

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